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# **Adult mortality and its impact on children in two informal settlements in Nairobi, Kenya**

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**7<sup>th</sup> January, 2013**

**Thesis submitted in fulfilment of the requirement for the award of the degree of Doctor of Philosophy (PhD).**

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## **Abstract**

This thesis examines the impact of adult deaths on children in two slums in Nairobi city. Over the last two decades, there has been a marked increase in adult mortality in Sub-Saharan Africa. Data on adult mortality in the region are scanty and this makes assessment of its impact on child well-being hard. The thesis analyses data from a longitudinal demographic surveillance system that monitors births, migration, and deaths and identifies causes of death using verbal autopsy. Other data collected include: household characteristics, schooling and health care utilisation. It investigates: i) levels, trends and causes of adult deaths; ii) the impact of adult deaths on children's household circumstances, and iii) the impact of adult deaths on children's health and social outcomes. Measures of adult mortality were estimated using life-table and survival analysis techniques. Regression techniques were used to assess impact of adult death on children's migration, living arrangements, survival, immunisation and schooling.

Life expectancy in the two slum populations was low. Adult mortality was higher in women than men. Ethnicity, gender, wealth status were associated with the risk of adult death. Overall, HIV/AIDS was the leading cause of adult death, followed by injuries and tuberculosis. The risk of death from HIV/AIDS was highest in Korogocho slum and the Luo ethnic group. Child mobility in the slums was high. After death of a mother, the risk of child out-migration increased. Death of a father increased average household size while death of a mother resulted in a reduction in household size. Households that experienced adult deaths were more likely to be headed by an older person. Death of a mother, especially from HIV/AIDS, but not that of a father, increased the risk of child death. The risk was highest in the 6 months before and after maternal death. The effect of adult deaths on children's education depended on slum of residence. While Viwandani children had better educational outcomes overall, death of a mother in Viwandani resulted into poorer schooling outcomes.

Interventions aimed at the leading causes of adult deaths need to be scaled up. The results here confirm that adult deaths negatively impact child well-being in this urban setting. Child survival can benefit from scaling up existing interventions, while mitigation of social impacts may require a mix of family and institution-based support for orphaned and vulnerable children.

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## List of Acronyms

AIDS	Acquired Immune Deficiency Syndrome
APHRC	African Population and Health Research Center
ART	Anti-Retroviral Therapy
CBS	Central Bureau of Statistics (Kenya)
DHS	Demographic and Health Survey
DSS	Demographic Surveillance System
HIV	Human Immunodeficiency Virus
ICD-10	International Classification of Diseases-10 <sup>th</sup> Revision
IMR	Infant Mortality Rate
INDEPTH	International Network for Demographic Evaluation of Populations and Their Health
MDG	Millennium Development Goals
MMR	Maternal Mortality Ratio
NUHDSS	Nairobi Urban Health and Demographic Surveillance System
ORT	Oral Rehydration Therapy
OVC	Orphaned and Vulnerable Children
U5MR	Under-five Mortality Rate
UN	United Nations
UNAIDS	Joint United Nations programme on HIV/AIDS
UNICEF	United Nations Children and Education Fund
UNFPA	United Nations Population Fund
UNESCO	United Nations Educational, Scientific and Cultural Organisation
YLL	Years of Life Lost due to premature death
WHO	World Health Organisation

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# 1. Introduction

This thesis examines adult mortality and the consequences of adult deaths in their households for children in two slum communities in Nairobi city, Kenya. In this initial chapter, I provide a general introduction to the problem of adult mortality, the challenges involved in its measurement and its impact on children in sub-Saharan Africa and in Kenya in particular. The chapter also outlines the aims of the study and its specific objectives and provides a brief summary of the study setting. Lastly, it provides a brief description of the contents of each of the subsequent chapters.

The central motivation for this thesis is that adult mortality in the study population is likely to be high given the findings of previous studies on HIV prevalence and the burden of disease profile <sup>[1, 2]</sup>. However, the exact burden of adult mortality and the major causes of death are not clearly known. Moreover, given the prevailing poor social and economic circumstances in this population, it is likely that children are highly impacted by adult deaths. Elsewhere, several studies have examined adult mortality and its impact on households, but a lot of emphasis has been put on examining impact due HIV/AIDS related adult deaths although other causes are also important <sup>[3, 4, 5, 6, 7]</sup>. Here, I examine impacts from all-causes but also look at specific causes. I examine impact on children because children are the most vulnerable members of households and likely to suffer indirect and direct effects of parental poor health, including vertical transmission of HIV, and consequences of parental death. Additionally, there is interest in unravelling the cause of the stall in the decline in child mortality in the recent years in Kenya and other parts of the region, which is suspected to be partly attributable to HIV/AIDS. This is yet to be confirmed.

In the last two decades, sub-Saharan Africa has experienced a marked increase in adult mortality <sup>[4, 8]</sup>. There is, however, a shortage of data on adult deaths in developing countries due to the lack of, or ineffective, vital registration systems. In Kenya, the proportion of deaths registered is estimated to be about 47% and details on age and cause of death are largely lacking, hence limiting the usefulness of the data <sup>[9]</sup>. Alternative data sources and analytical methodologies are therefore crucial to bridge the gap in adult mortality data. Surveys, censuses, sample registration, and small area

demographic surveillance systems (DSS) can all provide some limited mortality data on adults. DSS provide additional data on causes of death collected using verbal autopsies. Details of the strengths and limitations of these methodologies are discussed in chapter 2.

While data on causes of adult death are scanty, the available data show that HIV/AIDS is a leading cause of adult death. Other important causes include resurgent tuberculosis, and emerging non-communicable diseases and injuries. Different regions and countries in Africa are at different stages of the HIV/AIDS epidemic with different levels of infection in the general population <sup>[10, 11]</sup>. There are also within-country variations along social, cultural and demographic lines. In Kenya, HIV is reported to be more prevalent among women, adults, certain ethnic groups and in urban areas <sup>[2, 12, 13]</sup>. For example, whereas the prevalence of HIV is above 15% in Nyanza province and 10% in Nairobi, it is barely 1% in North Eastern province <sup>[14]</sup>. A recent study of HIV prevalence at the Nairobi Urban Health and Demographic Surveillance System (NUHDSS) site where this research was conducted, which is located in Nairobi province, showed the prevalence to be 12%, much higher than 6% for non-slum urban and 5% for rural areas <sup>[2]</sup>. Similarly, malaria is endemic in the Western and Coastal parts of Kenya and is the main cause of mortality among children. However, a recent study showed that malaria transmission in Nairobi is negligible or non-existent, even though many fever episodes are treated as malaria <sup>[15]</sup>. The variation in the levels and burden of major causes of deaths by socio-demographic characteristics and geographical location makes context very important in planning, implementation of interventions and resource allocation.

Because of the variability of HIV/AIDS epidemic, it is likely that the corresponding burden of adult deaths and their impact on communities, households and individuals also vary <sup>[16, 17, 18]</sup>. For example, in southern and eastern Africa where the HIV prevalence is high, the prevalence of orphanhood is correspondingly higher than in West Africa where the prevalence of HIV is much lower <sup>[16, 19, 20]</sup>. Death of an adult in a household may bring about changes in a household's social and economic situation. These changes in turn may impact children in several ways including compromised child care, reduced access to health care and nutrition, reduced survival and poorer social outcomes including educational attainment <sup>[5, 19, 21, 22, 23, 24, 25, 26]</sup>. The literature

further shows that the extent of impact of adult death depends on pre-existing circumstances such as the deceased person's role in income generation, contribution to care for dependants, gender, their household's wealth, and the composition of surviving household members <sup>[27, 28, 29]</sup>. It has also been noted that responses to adult deaths at household and community levels may influence short and long terms impacts on individuals and households. Responses to mitigate impact of HIV/AIDS vary and may not be necessarily successful, but may involve surviving household members taking on extra roles in the household like income generation, new members joining the household, and others perhaps leaving to ease stress on the limited resources <sup>[30, 31, 32]</sup>. In many parts of sub-Saharan Africa, the extended family plays a big role in taking care of orphans and several authors argue that this has helped cushion potential negative impacts, particularly on HIV/AIDS orphans <sup>[33, 34, 35]</sup>.

Although the number of studies that have been able to assess the impact of adult death longitudinally has grown over the last two decades, most have been based on rural populations <sup>[16, 22]</sup>. There is dearth of research on urban populations although rural and urban populations may differ systematically in ways that translate into varying levels of risk of adult death and varying responses to its impact. Sub-Saharan Africa is urbanising at a very fast rate but without commensurate economic and physical infrastructure development <sup>[36]</sup>. As a result, many urban residents live in slum settlements with limited means of livelihood, and limited access to medical and social amenities. Furthermore, while this sub-population is sizeable, they largely remain "invisible" to the authorities. The informal nature of the settlements makes planning and service delivery hard or enables it to be conveniently neglected. Correspondingly, the social and health outcomes of slums residents tend to be worse than those of non-slum urban residents to start with <sup>[2, 37, 38, 39]</sup>.

The impact of adult death on children in affected households may be mediated through loss of income to the households; poorer care; worsening provision of essentials such as shelter; lack of health care; inadequate nutrition and reduced emotional support. However, this thesis limits its assessment of impact in the following areas:



*Migration:* The study population is known to be relatively mobile. Often children migrate when their guardians do or in response to economic or demographic shocks. Migration may have negative or positive consequences for children.

*Living arrangements:* Children's living arrangements refer to children's circumstances in the household in terms of relations with other household members, size of household and household headship, among other things. Migration intimately influences the household structure and therefore who the child lives with. This is important as it has implications for the care, financial support, nutrition and health of a child.

*Child survival:* Adult death, particularly parental death, may mean that child care, nutrition, health care and safety are compromised and this might manifest as an increased risk of death for children who lose an adult in their household.

*Health care seeking, with vaccination as an example:* Death of an adult may compromise access to child health care due to lack of finances, or appropriate carers. Non-use of child health care services, particularly immunisation, may result in increased vulnerability of children to ill-health and raise their risk of death.

*Education:* Children's education is central to human capital formation and socialisation. Death of an adult may result into poor schooling due to loss of income, care, moral support and supervision to ensure that children attend school.

## **1.1 Definition of key concepts:**

**Adult death:** In this thesis, an adult death is defined as a death of an individual aged 15 years or more. Conventionally an adult is defined as an individual aged 18 years and above. For various reasons, different cut-offs have been used in various research projects. For example, most research on orphans and other vulnerable children use the age category 0 to 14 to define children, while studies on child labour uses an upper limit of 17 years. One of the key objectives of this work is the estimation of adult mortality. Adult mortality is usually defined as the conditional probability of death of an individual 15 years or older before reaching their 60<sup>th</sup> birthday. Thus, I maintained the age 15 as a cut off for defining adults through all the analyses as a mutually exclusive dichotomy of "child" and "adult" is needed. This is discussed further in chapter 3.

**Impact of adult death:** The impact of an adult death on a household and its members refers to any of the multidimensional changes that may result from and are attributable

to the event of an adult death in household. These may include economic changes (through reduced earnings, increased expenditure); changes in household membership and relations therein; changes in access to essentials such as food, health and health care; changes in roles of household members; and changes in participation in desirable social activities such as schooling. These changes are often judged against circumstances prior to an adult death or against members from households that did not experience an adult death. Some changes are easier to measure than others. For practical reasons, the assessment of adult death impact is limited to that of co-resident children irrespective of their social or biological relation to the deceased adult.

## **1.2 Study setting**

This thesis is based on longitudinal data derived from the Nairobi Urban Health and Demographic Surveillance System (NUHDSS), which has been operational since August 2002 on the outskirts of Nairobi city. The surveillance area covers two informal settlements with generally poor and relatively mobile people, the majority being rural-urban migrants. Between 60,000-70000 people are under surveillance at any given time and their vital events are recorded and updated every 4 months. Verbal autopsies are conducted when a death occurs to establish the probable cause of death.

The slum settlements in Nairobi city are own-built temporary structures on unused public land with no proper physical infrastructure. The informal settlements are underserved in terms of social amenities and have limited economic opportunities compared with up-market suburbs within the same city. Nairobi city has an estimated population of 3.1 million people with estimates indicating that more than 50% of the residents live in slum settlements<sup>[40]</sup>. The two slums where the NUHDSS is conducted represent a small fraction of the 50 or so informal settlements in Nairobi city.

As of the 2009 national census, the population of Kenya stood at 38.6 million people with 32% of them living in urban areas<sup>[40]</sup>. The cities and slum populations are an amalgam of individuals from the divergent backgrounds. Their characteristics to some extent reflect those of the diverse ethnic, social and economic backgrounds from which their inhabitants come since many of the slum dwellers migrated to the cities in adulthood. Nationally, there are 42 ethnic groups in Kenya with widely varying

population sizes and cultures. Of the 42 tribes, majority are either Bantu or Nilotic. The Kikuyu from Central region are the largest group. They are matrilineal, practice universal circumcision for men and economically more empowered than other ethnic groups. On the other hand, the Luo, another large ethnic group from the Western region, are a patrilineal ethnic group who do not traditionally circumcise men. Ethnic groups originating from the more arable and densely populated Western, Nyanza, Rift valley and Central provinces are traditionally farmers while those from semi-arid and sparsely populated North Eastern regions are semi-nomadic pastoralists.

Administratively, Kenya is divided into 8 provinces with several districts in each province. However, with the new devolved administration coming into force in 2013, this will be replaced by 47 administrative Counties with Nairobi province becoming Nairobi County.

A more detailed description of the study setting and operations of the surveillance system are provided in chapter 3.

### **1.3 Rationale, aims and objectives**

#### **Rationale**

Accurate data on adult mortality, particularly cause-specific mortality data are rare. Not only is it important to monitor patterns and causes of adult mortality, it is also important to understand its impact. Neither censuses nor national surveys, which are the main sources of demographic data in sub-Saharan Africa, collect cause-of-death data. Data from demographic surveillance systems (DSS) make it possible to measure adult mortality and its impact on households and individuals. To date, no comprehensive study has been carried out in Kenya using a longitudinal framework to assess the levels and impact of adult deaths in an urban setting. Previous studies have been mainly rural based and yet the distribution of major causes of adult deaths varies across the region. In addition, the urban population is growing at unprecedented rates of which slums account for two-thirds of urban population and yet there is no disaggregation for this population.

Lastly, the ongoing scale-up of life prolonging anti-retroviral drugs, which are expected to have major impact on adult mortality, emphasises the need to measure adult mortality at a population level. The health care sector, civil registration and ARV programs do not have the capacity to measure changes in adult mortality over time at a population level. Thus, estimating the impact this intervention is having on the population is difficult. The results from this study may both provide information on the initial impact of ARV treatment but also provide some insights on the trends in adult mortality attributable to HIV/AIDS.

### **Aims and Objectives**

Aim 1: To describe the level, trend and causes of adult mortality in two urban informal settlements in Nairobi.

#### **Specific objectives for aim 1:**

- i) To estimate adult mortality levels, trends, age patterns and compare life expectancy with data from other African sites. In addition, factors associated with the risk of adult death are assessed.
- ii) To establish major causes of adult death and estimate cause-specific adult mortality rates. Also, factors associated with the risk of adult death from specific causes are assessed.

Aim 2: To assess the impact of adult mortality on children's household circumstances including migration and living arrangements.

*Hypothesis 1:* Children from households with an adult death are more likely to migrate than children from households with no adult death.

*Hypothesis 2:* Children from households with an adult death are likely to live in larger and poorer households than children from households with no adult death.

#### **Specific objectives for aim 2:**

- i) To estimate the excess risk of child migration from households with an adult death compared with that from households with no adult death. Both out-migration from the two slums and internal change of place of residence (internal migration) are considered.

- ii) To describe children's living arrangements including household size and the household head's age and how this is impacted by an adult death in household.

Aim 3: To assess the impact of adult mortality on child health and educational outcomes.

*Hypothesis 3:* Children from households with an adult death have lower survival than those from households with no adult death.

*Hypothesis 4:* Children from households with an adult death are less likely to be fully vaccinated than those from households with no adult death.

*Hypothesis 5:* Children from households with an adult death are more likely to have poor schooling outcomes than those from households with no adult death.

### **Specific objectives for aim 3**

- i) To estimate the excess risk of death, if any, of children from households that experienced an adult death.
- ii) To describe specific causes of child deaths and estimate risk of death from the specific causes.
- iii) To describe childhood immunisation status by adult mortality status in the household and estimate the risk of incomplete or untimely vaccination.
- iii) To examine school participation of children aged 6-14 years from households with exposure of adult deaths vis-à-vis those with no adult deaths and identify factors associated with poor educational outcomes.

## **1.4 Structure of thesis**

The following 11 chapters comprise a literature review, a discussion of data and methods, eight results chapters, and an overall discussion chapter. Each of the eight results chapters has a short background section, a methods section detailing matters that are specific to the research question in the chapter, and results and discussion sections.

Chapter 2 provides a critical review of the relevant literature highlighting current knowledge, contentious issues, knowledge gaps and potential research areas. One section is dedicated to describing sources of data on adult mortality, levels and trends in

adult mortality, measurement challenges and causes of adult deaths. The rest of the chapter covers the impact of adult death on children's social and health outcomes with a focus on child migration and living arrangements, child survival, immunisation and educational attainment.

Chapter 3 describes the methods used in data collection, management and analysis. A detailed description of the surveillance system and the population under surveillance is given. Data preparation and various analytical methodologies used are discussed. Further details on specific analytical procedures are discussed in the chapter-specific methods sections.

Chapters 4 and 5 present results for all-cause and cause-specific adult mortality respectively. Chapter 6 examines child migration patterns both within and without the informal settlements. Chapter 7 examines children's living arrangements and how they are affected by an adult death in a household. Chapter 8 examines all-cause child mortality levels and trends and how these may be influenced by adult death in households. Chapter 9 discusses the major causes of child mortality and the impact of adult deaths on child mortality from specific causes. Chapter 10 and 11 presents the results concerning the impact of adult deaths on child health care utilisation (immunisation) and educational outcomes respectively.

The last chapter (12) discusses the findings of the thesis and puts the results in perspective, offering explanations, contrasting the results presented here with those of other studies, highlighting limitations, and identifying policy issues and potential areas for future research.

## **2. Literature review**

### **2.1 Introduction**

This chapter provides a concise review of current state of adult mortality in sub-Saharan Africa and its impact on children. The review is organised by theme identifying areas of contention, agreement, methodological challenges, gaps and research opportunities, some of which informed the research questions posed in this study.

The first section of the literature review tackles issues around the challenges of measuring levels and causes of adult mortality. First, I examined the literature on levels of adult mortality in the region and Kenya. Next, the literature on data sources for adult mortality, methodological and analytical challenges and developments in estimating adult mortality is reviewed. Lastly, I reviewed the literature on factors associated with all-cause adult death; and the major causes of adult death and associated factors.

The second major theme explored is the literature on the impact of adult deaths on children. The first subsection looks at the literature on the impact of adult death on children's migration. This is followed by the review of the literature on impact of adult death on children's living arrangements. This sub-theme and the previous one are closely linked, as migration brings about changes to household membership composition. Next, I reviewed the literature on the impact of adult mortality on survival of children aged less than five years. The next sub-theme looks at the impact of adult death on child health care utilisation- focusing on child immunisation. The last sub-theme of the chapter looks at impact of adult death on educational outcomes for children between 6 and 14 years of age.

### **2.2 Measuring adult mortality: level and trends**

Data on adult mortality in the developing world are very scanty due to lack of vital registration which is the most credible source of mortality data <sup>[41, 42]</sup>. Setel et al. refers to the lack of vital registration “a scandal of invisibility” because many individuals in sub-Saharan Africa are born and die without any official trace or count <sup>[43]</sup>. In the sub-Saharan

region, with the exception of Mauritius, Seychelles and South Africa, and to a lesser extent Zimbabwe, there are hardly any adequate vital registration systems with few of those that exist registering more than 50% of all deaths but with poor or no recording of causes of death <sup>[42, 44]</sup>. Moreover, this deficiency is happening in the region with the highest estimated adult mortality rates globally. In Kenya, about 50% of deaths are registered but causes of death are not systematically recorded and reported on and other important information such as age are missing or inaccurately recorded <sup>[9, 42]</sup>. Furthermore, not much analysis on adult mortality has been carried out using these existing data. Even with a functioning vital registration system, assessment of the impact of adult deaths on households and their members requires a community-based study as data collected by the vital registration system is limited and not linkable to household characteristics.

Due to the lack of vital statistics, adult mortality estimates for most of Africa have been obtained mainly by indirect estimation. They have been estimated either from under-five mortality using model life tables <sup>[45]</sup> or from surveys and census that have asked questions on the survivorship of siblings or parents. Indirect methods have worked well in estimating infant and under-five mortality, but they often perform poorly in estimating adult mortality <sup>[46]</sup>. Indirect methods usually make assumptions about the population and use data derived from various sources and collected using different methodologies. As a consequence, estimates are bound to vary.

National censuses can provide data on adult mortality where at least two consecutive censuses have been conducted. Where additional data on inter-censal mortality patterns are available, for example from surveys, these can be adjusted for under reporting using the estimates derived from the census data. Census data, while widely used to estimate adult mortality, have a number of limitations including being infrequent and thus not suitable for understanding recent trends in adult mortality; estimates derived are also known to be sensitive to differences in census coverage and inaccurate reporting of age. Additionally, census data do not have information on causes of death, and are therefore not useful in studying impact of death from specific causes on households and individuals. Lastly, assumptions made in the methodologies such as the census survival, growth balance and



extinct generation methods of estimating adult mortality are based on assumptions that may not always hold. The assumption of a closed population or one with minimal migration makes it hard to estimate adult mortality for populations that exhibit high levels of migration. In addition to the assumption of the population being closed, the methods also assume that the inter-censal population experiences minimal changes in mortality and fertility when these actually are likely to change during the interval <sup>[45]</sup>. Kenya routinely conducts national censuses every 10 years with some coverage problems in the semi-nomadic, insecure and arid northern parts of the country.

The other group of methods for the indirect estimation of adult mortality uses data from either surveys or censuses and inquires about the survival of relatives such as parents or siblings <sup>[45, 47]</sup>. The great advantage of these set of methods is that they do not require that the population be closed to migration. As such, these methods can be used to estimate adult mortality on subpopulations. The downside of the survey or census data used in these methodologies is that the surveys and censuses are infrequent and thus unable to provide good trend measures over short periods of time. They also suffer from recall bias and age mis-reporting of parental or sibling survival and ages, particularly, for deaths that occurred in infancy or early childhood. Due to the inherent problem of recall and age misreporting in surveys, it has been observed that figures from surveys are underestimates of prevailing adult mortality levels <sup>[48]</sup>. The Demographic and Health Surveys (DHS) currently being implemented in many developing countries collect a full sibling survival history. Using life table approach or indirect method using proportion of surviving siblings, adult mortality can be estimated <sup>[8, 48]</sup>. These data, however, lack information on causes of death and are also subject to survival and recall bias and the adult mortality estimates are believed to be downward biased <sup>[46]</sup>. Comparing adult mortality estimates with UN estimates, Gakidou et al. noted that there was good correlation between the estimates from the two sources but also noted that there was likely under reporting of mortality in surveys <sup>[44]</sup>. Nevertheless, given that vital registration systems in the developing world are not keeping pace with the need for adult mortality data, surveys and censuses remain important sources of adult mortality data.

A variety of model life tables have been developed and used in estimating and understanding adult mortality where no mortality data exist <sup>[49, 50]</sup>. Model life tables are based on the fact that there are predictable similarities in age-patterns of mortality between populations. Thus, reliably measured mortality estimates at a given age can be used to predict mortality in another age group using a mathematical relationship. In this way, mortality estimates for the entire population can be derived using a limited amount of data, for example under-five mortality and a standard model life table. One limitation of the model life tables that are in use is that they were not derived from empirical African population data. It is possible that mortality age patterns in the region are distinctive or have changed since these model life tables were developed. Therefore, the assumption that mortality in sub-Saharan Africa conforms to any of the available models fitted to a given estimate of under-five mortality rate may not be accurate <sup>[41]</sup>. Indeed, adult mortality estimates derived this way tend to be lower than expected, particularly in populations affected by HIV/AIDS <sup>[51, 52]</sup>. Estimates from WHO, accounting for HIV/AIDS show that in most countries in Sub-Saharan Africa, the probability of an individual aged 15 dying before reaching the age of 60 was about 50% which is in sharp contrast with the developed world <sup>[53]</sup>. More recently, the INDEPTH network published model life tables based on surveillance data drawn mainly from rural areas. These tables are yet to be widely used <sup>[54]</sup>.

Recent years have seen the establishment of several population-based longitudinal health and demographic surveillance systems (DSS). These DSS collect data on births, deaths and causes of death using verbal autopsies. The DSS is an open cohort whereby individuals from the initial census can leave the surveillance area while others can enter surveillance along the way. While still under surveillance, information on individuals is collected and updated at regular intervals. Surveillance systems are, however, labour-intensive and expensive to maintain and as such they are normally limited in size of the population that they follow up. This limits the generalisability of DSS data <sup>[55]</sup>. The coverage issue has been addressed, to some extent, by countries having multiple surveillance sites which sometimes cover both rural and urban areas <sup>[55, 56]</sup>. Surveillance sites under the INDEPTH umbrella have pooled mortality data to estimate mortality in the sub-Saharan region and, indeed, model life tables based on these data have been published <sup>[54]</sup>. Just like the UN

derived estimates of adult mortality, the INDEPTH data showed that adult mortality is higher among men and higher in the southern and eastern African region as compared with the West African region. It should, however, be noted that the demographic and health surveillance sites that contributed data to the INDEPTH publication were predominantly rural and therefore the two broad model families identified may not fit well mortality data from urban sub-Saharan African populations and this needs to be examined using urban DSS data <sup>[54]</sup>. While the methods for measuring adult mortality have been improving over time, most of them are based on retrospective data and this makes linking of adult mortality to other individual and household level outcomes difficult. On the other hand, DSS data are longitudinal and adult mortality can easily be linked to other household and individual outcomes. Thus, the longitudinal nature of the data allows for examining the later consequences of adult death for children. This makes these data rich in content.

### **2.2.1 Adult mortality levels and trends**

In countries severely affected by HIV/AIDS, adult mortality has risen over the last two decades reversing trends observed earlier <sup>[8, 57, 58, 59]</sup>. Studies across sub-Saharan Africa show variations in adult mortality levels. Some of the variation could be explained by the differences in the underlying level of HIV prevalence as HIV is a leading cause of adult death <sup>[58]</sup>. The differences in adult mortality between places could also be due differences in access to and utilisation of life prolonging anti-retroviral therapy. In countries with widespread social strife such as civil war, many lives of adults have been lost directly due to war but also because war fuels the spread of HIV. Historically women, wealthier individuals and urban residents have had a survival advantage <sup>[60]</sup>. However, with the spread of HIV, which disproportionately affects women, evidence is emerging that shows that the mortality gap is narrowing or reversing. For example, it has been shown that women aged 24-39 are experiencing higher mortality than men <sup>[8, 57, 61]</sup>. The KDHS 2003, observed a higher adult mortality rate and more marked increase since the previous survey estimates for women than men. This was partly attributed to a disproportionately higher prevalence of HIV among women in Kenya <sup>[12]</sup>. In the subsequent survey of 2008/9, adult mortality among females was slightly lower than that of males but there was a significant

crossover between the ages 20-39 years with women having a higher adult mortality in this age group. However, while the sibling survivorship approach used by DHS provides some idea of the situation regarding adult mortality, it is only based on small samples and subject to recall bias, especially with regard to sibling age reporting.

Estimates of adult mortality for Kenya as estimated by WHO show that the probability of dying between 15 years and 60 years ( $_{45}q_{15}$ ) was 48% for men and 50% for women in 2004<sup>[62]</sup>. More recent estimates show that adult mortality was 25% for women and 33.5% for men in 2010<sup>[53]</sup>. In addition to HIV/AIDS and given the poor state of health care infrastructure, obstetric complications and maternal death significantly contribute to adult mortality among women. This is evidenced in the poor progress being made in Kenya on Millennium Development Goal 5 (Reducing the Maternal Mortality Ratio by three quarters by 2015). In 2003 the maternal mortality ratio for Kenya was estimated at 414 deaths per 100,000 live births, compared to 488 per 100,000 live births in 2008/9<sup>[12, 13]</sup>.

Unfortunately, the sample size in the KDHS is normally too small to allow for regional estimates or urban-rural delineations.

### **2.2.2 Causes of adult death**

The developing world is undergoing a delayed epidemiologic transition whereby parasitic and infectious diseases and under nutrition give way to non-communicable diseases with declining overall mortality<sup>[63]</sup>. While the epidemiologic transition theory provides a general framework for understanding of how mortality and cause of death have influenced population changes over time, this generalisation may have exceptions. The theory did not highlight or anticipate many issues that are unfolding and influencing mortality and population growth in sub-Saharan Africa. For example, the role of poverty and disease risk; and the relative importance of the various causes of death are not accounted for. Also the issue of emerging and re-emerging infectious diseases is critical to current and future mortality experience even in populations whose transition is following the expected trajectory or that have completed the transition.

Sub-Saharan African populations are not homogenous. While some countries have had a slow but steady increase in life expectancy, others have had stalls or reversals, some of these have related to economic crises, others to the coming on the scene of the HIV/AIDS epidemic, and yet others to both developments <sup>[64, 65, 66]</sup>. Currently sub-Saharan Africa exhibits both spectra of disease in substantial measure. While the burden of non-communicable diseases is growing at unprecedented rates, especially in urban populations, parasitic and infectious diseases remain a major cause of ill-health and death in many parts of Africa, while new and re-emerging diseases have come on the horizon <sup>[67]</sup>. In rural Senegal, a study found that 51%, 32% and 17% of deaths were accounted for by non-communicable diseases, injuries and communicable diseases respectively among men. The corresponding estimates for women were 35%, 49% and 16% for non-communicable diseases, injuries and communicable/reproductive causes respectively <sup>[68]</sup>.

Nationally-representative data on causes of death are not readily available for most countries in sub-Saharan Africa due to the lack of functional vital registration systems. Even where partial vital registration exists, cause of death information is largely missing or not appropriately coded to a standardised system- the International Classification of Diseases (ICD)-so as to allow for cross comparison. Medical records and the entire Health Management Information System (HMIS) are also poorly managed and incomplete as most people die outside of the formal health facilities. Vital registration has both a legal and statistical use. In Kenya, by law, burial permits are required by authorities and this explains the fact that about 50% of deaths in Kenya are registered. However, because the emphasis is put on the legal dimension rather than the statistical one, information such as age and cause of death is often not recorded accurately, rendering the data of limited use.

### **2.2.3 The verbal autopsy**

Owing to the challenge of the lack of data on causes of death, alternative sources of cause-of-death data have been devised based on both health facilities and community-based studies <sup>[69, 70]</sup>. Data from relatively small community-based studies across the region have provided insights on the major causes of death <sup>[1, 4, 71, 72]</sup>. Detailed account of illness of the

deceased are taken and evaluated by a group of clinicians or computer algorithms to try and ascertain the most probable cause of death <sup>[73, 74]</sup>. This approach is often referred to as the “Verbal Autopsy”. As contrasted with the conventional autopsies, the narratives given about the illness and medication administered before death are used to ascertain the cause of death. The World Health Organisation has spearheaded development, standardisation and harmonisation of verbal autopsy tool to increase the validity and comparability of the data collected across various surveillance sites but variations in the tools being used in different places remain <sup>[75]</sup>.

While much has been achieved by means of verbal autopsies, the approach has limitations. Field workers who conduct the interviews may misrepresent symptoms and the misrepresented information may lead to wrong diagnoses. At the coding level, the coders may be biased towards assigning particular causes and not others given their local knowledge of prevalent diseases. As a result, the verbal autopsy tool is known to have limited sensitivity and/or specificity especially for conditions that present with non-specific symptoms, for example HIV/AIDS, cardiovascular diseases and malaria <sup>[76, 77]</sup>. For other diseases such as measles, injuries, and maternal causes, the sensitivity and specificity are fairly good <sup>[77, 78]</sup>. In spite of these concerns, the approach has been successfully used in many countries in sub-Saharan Africa to measure mortality in both adults and children. Currently, most DSS sites conduct verbal autopsies to ascertain causes of death including the NUHDSS site, which generated the data used in this thesis. In practice, in spite of its limitations, this approach has been found to reasonably identify causes of death of public health importance in many sub-populations and it is continually being improved upon.

In 2011, the World Health Organisation published a standard VA tool that has been developed to help improve comparability across the board <sup>[75]</sup>. Ascertainment of probable cause of death from the collected data however remains a big challenge. The commonly used method-the physician-led process is costly, slow and generates results that are not highly reproducible within and between physicians and also not fully comparable across populations <sup>[79]</sup>. In recent years, efforts have been made to computerise assignment of cause-of-death using probabilistic models. One such tool that is in the public domain is the

probabilistic model for interpreting verbal autopsies known as InterVA <sup>[80, 81]</sup>. Initial results on the performance of InterVA tool showed low performance relative to other methods <sup>[73, 82]</sup>. However, recent revisions of the tool are encouraging, with improved comparability and reproducibility of results across sites. It also has the additional appeal of reducing the cost and time involved in assigning causes of death compared with a physician-led process <sup>[80, 83, 84, 85]</sup>.

While initial studies on the performance of physician and algorithm-led coding of causes of death focused on the validity of the process, some argue that developing a system that can provide reproducible and comparable results across the board is more important than focusing on the validity of the approach, particularly as the “gold standard” (hospital-based medically certified deaths) is itself not without fault, partly because such data are not representative of the population <sup>[83, 84]</sup>. The argument has been advanced that the limitations of the VA approach are well known and that, as long as the results are interpreted as of public health importance rather than clinical significance, that is good enough <sup>[83, 86]</sup>. While this might be true to some extent, the desire to have valid results cannot be totally ignored in favour of cutting costs, time and achieving high reproducibility: some degree of validity is required for the results to be of any value.

Other sources of cause-of-death data include medical records in health facilities. Although data from medical records are often incomplete and not standardised, they provide important insights in cause of death among facility-based deaths. Some of these records (medical autopsy) have been used to validate verbal autopsy diagnoses <sup>[76, 77]</sup>. These data, however, are of limited use for understanding the distribution of major causes of death at population level.

In countries with generalised HIV epidemics in sub-Saharan Africa, current evidence shows that HIV/AIDS is the leading cause of death among adults and is responsible for the marked reduction in life expectancy in many countries in the region <sup>[71, 87, 88, 89, 90, 91]</sup>. Preliminary research in the slums of Nairobi showed that HIV/AIDS combined with tuberculosis accounted for about 50% of years of life lost (YLL) due to premature death

among people five years and older <sup>[1]</sup>. Another study in rural area in Kenya with a high prevalence of HIV infection showed that HIV/AIDS and tuberculosis accounted for about 48% of all deaths among children and adults <sup>[71]</sup>. These studies, however, did not provide the distribution of major causes of adult deaths separately. However, the results imply that the proportion of adults dying of HIV/AIDS is even higher than that reported for the whole populations since HIV prevalence is higher among adults than children.

The HIV/AIDS epidemic has caused a resurgence of other infectious diseases, particularly tuberculosis. In 2007 it was estimated that of the over 33 million individuals infected with HIV, about a third of them were co-infected with tuberculosis <sup>[92]</sup>. The prevalence of tuberculosis has more than doubled over the last two decades in countries with a high prevalence of HIV and it is a major cause of death among HIV infected individuals <sup>[92]</sup>. Tuberculosis is highly infectious but treatable if it is detected early and the treatment regimen is adhered to. The case-detection rate in Kenya is relatively low and a sizeable proportion of patients do not adhere to treatment <sup>[93, 94]</sup>. This has resulted in the development of multi-drug resistant tuberculosis strains whose treatment is more difficult and expensive <sup>[95]</sup>.

Among women, maternal mortality ratios remain high, being estimated at about 488 maternal deaths per 100,000 live births in Kenya <sup>[13]</sup>. Among all the MDGs, the MDG on improving maternal health and the target of reducing maternal mortality ratio by three quarters is one of those on which least progress has been seen in Kenya and other African countries. The maternal mortality ratio in 2003 was estimated at 414 per 100,000 live births and in 2008/9 it was estimated at 488 per 100,000 live births <sup>[12, 13]</sup>. Typically maternal mortality is difficult to measure even in surveys given the fact that it is a rare event and thus require large sample sizes of women in the survey. Also, just about 43% of pregnant women deliver in health care facilities and thus many maternal deaths are never captured by the formal HMIS system <sup>[13]</sup>. The scanty available data, however, indicate that delivery-associated complications account for a sizeable proportion of adult deaths. Additionally, unsafe abortions contribute substantially to maternal deaths, currently estimated at about



30%, though this figure comes from a small study with small number of maternal deaths [96].

Non-communicable diseases and external causes of death are on the increase in certain populations in sub-Saharan Africa. A study in rural South Africa showed that diabetes and cardiovascular diseases were important causes of adult death in a population predominantly afflicted by HIV/AIDS [97]. In Mlomp, a rural population surveillance site in Senegal, assessment of causes of adult death showed that non-communicable diseases such as cancers were the predominant cause of adult death (though HIV/AIDS was not a major cause of death which is not the case in eastern and southern Africa) [68]. A study in the NUHDSS site (slums) showed that external causes accounted for about 18% of years of life lost due to premature death among people 5 years and older [1]. Another study in the same population showed that interpersonal violence was common, especially among young men, accounting for about 40% of all deaths from injuries. Road traffic crashes are another important cause of adult death.

### **2.3 Impact of adult mortality on children**

Adult deaths account for a substantial number of years of life lost (YLL) due to premature death. Adult deaths come with consequences such as lost productivity and compromised wealth creation at both the national level and for affected households and individuals. This study limits itself to assessing the impact of adult deaths on children, focusing on health and social outcomes. While surviving adult household members might be impacted, the effects may not be as profound as that on children due to the latter's greater physical, biological and social vulnerability. Orphanhood may result into increased child mortality and hence higher years of life lost due to premature death. It may also reduce investment in children's development, which may reduce their future productivity and standard of living. Concomitantly, all this may have a ripple effect on other individuals, households and communities.

The literature review below examines the current state of knowledge about the migration, living arrangements, health-care seeking, survival and educational outcomes of children affected by adult deaths. The review also identifies gaps, limitations, methodological issues and potential future research. Issues identified here partly feed into the conceptual framework of the impact of adult death on children in poor urban and relatively mobile population that is developed in this thesis.

### **2.3.1 Impact of adult death: Child migration**

Following death of an adult, changes to accommodate the shock in the household might dictate that either new member(s) join or some existing members leave the household. Whereas such movements might benefit the individuals concerned, they may also have negative effects on other household members, such as compromised access to health care, schooling, nutrition, or proper housing among others <sup>[98]</sup>.

#### **Patterns of migration in Kenya**

Following independence, the restrictions imposed on rural-to-urban migration during the colonial period were relaxed and this saw an influx of migrants from rural to urban areas. Motivations for migration are generally conceptualised around pull and push factors. Push factors are those that operate in the area of origin to make one to move away to another place, while pull factors relate to the factors at the destination that act as incentives for one to move to it. The search for economic opportunities stands out as one of the main reasons for migration, particularly among adults, but other factors are also important, including better education, healthcare, housing, security, natural disasters and family ties among other incentives. In the postcolonial era, rural-to-urban migrants were predominantly men in search of jobs in cities. Increasingly, women are accessing education and competing for semi-skilled and skilled jobs and, as a consequence, many are migrating on their own and starting families as migrants in the city <sup>[99]</sup>. A recent study in the two slums in Nairobi studied here showed that there was more intense movement among women than men and that there was a positive association between migration and education <sup>[100]</sup>. Other women migrate either together with a migrating husband or alone to join a husband already living

in the city. Studies have shown that individuals who are already married and had children are less likely to migrate into the cities <sup>[100, 101]</sup>.

Over the years, the pace of urban population growth has outstripped the rate at which jobs and amenities such as housing, schools, and health care have grown. This has resulted into a huge urban poor population living in slums for prolonged periods of time. Many of the migrants to cities who live in the slums have little access to livelihoods and limited access to social amenities. Because of this, they tend to move from one place to another within and between slums. Many slum residents also maintain links with their rural kin and often travel and stay in their rural homes for variable durations before returning to the city. As a result, there is generally a high turnover of residents in slums. This circular form of migration has been documented in the two slums studied here <sup>[100]</sup>. It has been estimated that about 25% of residents in the slums migrate annually and that this rate is especially high among young adults aged 20-24 years <sup>[100]</sup>.

### **Child migration**

Because children tend to move with their parents as a family unit, or be left behind in rural areas when their parents migrate, child migration has not been the focus of much research and has hardly been examined in its own right. Often, when parents first move to urban areas, their children tend to be left in the rural home either with one of their parents or with grandparents or other relatives until such a time as when the family is established in the urban areas. Subsequently, children might join their parents in the towns to live with them and probably attend school. On the other hand, some children born in cities, especially to single parents, may be sent to rural areas to live with their grandparents or other relatives to attend cheaper schools and reduce the burden of providing care and other costs of living, which are often higher in the cities. Children might also migrate in response to economic and demographic shocks in their households. In situations of conflict, children and their guardians may be forced to migrate to safer places <sup>[102, 103]</sup>.

Child migration is becoming of increasing research interest for a number of reasons. First and foremost, more children are migrating unaccompanied by their parents for reasons

including schooling, care, or labour<sup>[104]</sup>. Secondly, such migration may result in unfavourable child's living arrangements and hence their health and social wellbeing. The emerging trend may reflect increasing economic difficulties in the sending communities. The worsening economic situation both in rural and urban areas, compounded by the growing burden of prime-age death of parents, mostly associated with the HIV/AIDS epidemic, are partly responsible for this. Before the advent of ARV for HIV/AIDS treatment, it was noted that there was an increase in ill-health associated migration from South African cities, which were generally more affected than rural areas, to rural areas by individuals who were terminally sick<sup>[105]</sup>. Motivations for this were loss of income due to poor health, the high cost of living in cities, and the need for personal care. Even when death of an adult occurs in the city, often the rest of the family members, including the children, relocate to rural areas or less expensive cities or where they can get the extended family support, especially if the dead adult was the primary bread winner for the family<sup>[105, 106, 107]</sup>.

### **Impact of adult death on child migration**

Due to scarcity of longitudinal data, few studies have assessed child migration and how it is related to adult death. However, available evidence shows that the impact of an adult death on child migration depends on: whether the deceased was the household head, the mother or someone else, on the nature of death (sudden or protracted illness), on household size, and on the child's characteristics<sup>[24, 108, 109, 110]</sup>. Hosegood et al. in their 2009 study opine that household dissolution and migration of members following an adult death are interlinked. However migration can also happen in the absence of household dissolution. They also underscore the fact that not all occurrences of household dissolution and subsequent migration of household members are negative, arguing that it might be a strategy to positively respond to the effects of lost income or carer occasioned by an adult death in the household<sup>[109]</sup>. Studies in Tanzania and South Africa examined the impact of adult mortality on migration and household dissolution and noted that death of an adult increased the risk of household dissolution<sup>[24, 110]</sup>. The adult who dies in the household influences the likelihood of household dissolution and or migration of household members. Parental death is more likely to result in child migration than non-parental death. This is

particularly the case if a mother died <sup>[108]</sup>. Similarly, death of a household head is more likely to result in household dissolution and migration of household members including children than other adult deaths <sup>[110]</sup>. In countries with marked levels of adult deaths due to HIV/AIDS, it has also been demonstrated that the risk of household dissolution increases with multiple adult deaths in a household <sup>[24]</sup>.

The nature of or cause of death has also been found to be associated with risk of household dissolution and migration of members from affected households. A study in South Africa showed that the likelihood of household dissolution markedly increased if one or more members of the household died from violent or accidental causes but not HIV/AIDS <sup>[24]</sup>. The opposite has been reported in other settings where HIV/AIDS deaths were associated with higher likelihood of household members migrating or the entire household dissolving <sup>[31]</sup>. While chronic and protracted illnesses such as HIV/AIDS deaths drain resources over time, such deaths are often anticipated. Thus, some remedial plans are probably drawn up for the household members. This may involve, for example, cutting down on expenditure, moving to a smaller and cheaper house, and withdrawing children from school. Migration might be the final mechanism of adjusting, aimed at moving to cheaper places or where the extended family can offer support. In contrast sudden deaths, for example from accidents or violence, do not allow time for households to plan and adjust and migration might be the easiest option. So, while both anticipated and sudden adult deaths might ultimately result in an increased risk of household dissolution and migration, the former does so less than the latter and, if it happened, the move is more likely to be planned while the sudden death it is unplanned. The consequences for the households' members including children in the aftermath in the two scenarios might be different.

### **Consequences of migration**

Research has shown that migration can have both positive and negative outcomes on migrants in terms of health and other socio-economic outcomes <sup>[111]</sup>. Often migration is an adjustment strategy in response to social and economic stresses or seeking of opportunities. While the reasons for migration might be well intentioned, migration might come with adverse consequences such as an increased risk of death, limited access to health care and

other amenities, as some rural areas are seriously underserved in terms of geographical access to health care facilities, and poorer quality of care <sup>[112, 113, 114, 115]</sup>. On the other hand, while child migration has been looked at from the negative side, child migration may be associated with benefits both to the child and the whole family. Children might migrate to destinations where they will be better looked after in terms of care, nutrition, safety, health care and schooling. Even for the much criticised child labour associated migration, some studies in Bangladeshi, Philippines and Ghana have shown some positives in older children migrating for labour especially if it is a family arranged placement with adequate social capital in the destination. Many of children look at themselves are responsible in making a contribution to their own well-being and that of the family <sup>[116, 117, 118, 119, 120]</sup>.

### **Theoretical framework**

The impact of migration on child health and social outcomes can be explained based on three theories that have been used to understand child survival among rural-urban migrants:

- i) The theory of disruption: Migration may disrupt social bonds, relationships, income, care, schooling, access to care, and nutrition among other things. The disruption may have adverse consequences for children's social and health outcomes such as increased mortality, low access to immunisation and health care, poor physical growth and poor education outcomes <sup>[114, 121, 122, 123]</sup>.
- ii) The theory of selection: This theory posits that the propensity for an individual to migrate is determined, to some extent, by their characteristics such as income, education, gender and other characteristics. In the case of child migration, there might be certain household and individual guardian/child characteristics that make them more likely migrate <sup>[124, 125]</sup>. This implies that migrants are a selected group who might possess traits that may result into different social and health outcomes upon migrating. However, while this might apply in case of rural-urban migration, individuals who migrate following an adult death are not self-selected and may not have the traits that rural-urban migrants seeking opportunities may possess.
- iii) The theory of adaptation: Migrant adaptation means that after the disruption brought about by migration, the migrant has to change to the new realities in his

or her new environment. This may include changes in access to sanitary services, economic opportunities, disease prevention, health-care seeking, nutrition, and education among others. The consequences of failure of migrants to adapt may include low access to health care, poor child survival, and poor access to education, food, shelter, and other amenities <sup>[114, 115, 126]</sup>.

In the context of the NUHDSS, our understanding of the consequences of out-migration is limited by the fact that out-migrants are not followed up to ascertain their outcomes. A recent study in the two slums studied here showed that child mortality was highest among children whose mothers recently migrated into the slums <sup>[122]</sup>. While food might be expensive in the urban areas, in some rural areas it might be totally unavailable, especially in areas prone to droughts, with the potential consequence of compromised child nutrition, growth and development. Also, there are many rural areas where malaria is endemic whereas transmission is low in Nairobi <sup>[127, 128]</sup>. Migration of non-immune children from Nairobi to such malaria endemic areas might pose an extra risk of severe malaria and associated complications. Change of residence through migration might mean living with totally new household members. This might have implications for child care, nutrition and health-care seeking. Also, separation from familiar family members such as parents and siblings might have a negative emotional impact on the child, resulting into poor child behavioural adaptation. While most of the literature points to negative impacts of migration to child well-being, as pointed out earlier, migration of orphaned and non-orphaned children might be beneficial to the child and his/her family <sup>[33, 119, 129, 130]</sup>.

### **2.3.1 Impact of adult death: children's living arrangements**

The importance of understanding children's living arrangements is informed by the desire to have children looked after by the most appropriate carers. The question to be answered in this section of the review is: does adult death substantially affect children's living arrangements in terms of the size of the household they subsequently live in, relationships in the household and characteristics of household head? Before examining the literature on the potential impact of adult death on children's living arrangements, I explore the literature

of children's living arrangements in general in sub-Saharan Africa and Kenya. Changes of the living arrangements occasioned by adult death in households need to be interpreted in light of the prevailing general patterns as these may be influenced by other cultural, economic and social circumstances in a given population.

Household structure in sub-Saharan African varies between countries and there are also rural-urban differences. In 1996, household size for households with children in urban areas in Kenya was 5.7 compared to 7 in rural areas. The same study showed other countries such as Senegal, Namibia, Cameroon, and Burkina Faso have larger household sizes. Kenya and other countries such as Rwanda, Madagascar and Nigeria have nuclear households as the predominant type while for countries like Zambia, Senegal, Cameroon, Namibia and Burkina Faso; extended households are the most prevalent. Kenya has a unique feature with 14% of children living in households with a single parent whereas this is less than 5% in most other African countries with the exception of South Africa<sup>[131, 132]</sup>. The average household size in the two slums in Nairobi is less than 3 people<sup>[133]</sup>.

### **Impact of adult death on household structure**

Whereas death of an adult reduces the number of household members by one, rearrangements might occur whereby other members either migrate into or migrate out of the affected households. Sending away household members from an affected household might be aimed at cutting costs for the household. In the case of child out-migration, the motivation might be care related, in which case children might be sent to grandparents or other suitable carers. New members might join affected households to boost income generation, for marriage or to provide domestic support. A multi-country study in Africa showed that overall net household sizes reduce following death of an adult<sup>[134]</sup>. Another study conducted in rural South Africa showed that household size decreased and the proportion of female-headed households increased corresponding to a period of increased adult death due to HIV/AIDS<sup>[135]</sup>. In spite of the changes observed above, another study in the same rural population noted that the extended family structure still persists<sup>[35]</sup>. A survey carried out in rural Kenya showed that household size decreased by about 0.61



member following death of an adult, with more than a one person decrease if the deceased adult was the household head or spouse<sup>[27]</sup>.

In countries adversely affected by HIV/AIDS, the prevalence of orphans has dramatically increased over the past two decades<sup>[16, 18, 25, 135]</sup>. Although it is common practice in sub-Saharan Africa for non-orphaned children to live with adults other than their parents (voluntary fostering), death of a parent tend to result into a child living with relatives, as an obligation, who are sometimes not in a position to care and provide adequately for the orphan (crisis fostering). Whereas non-orphaned children who do not live with their parents may receive remittances from their non-resident parents, this is not the case with orphans. Also, while single orphans, particularly maternal orphans, may get financial support from their surviving parent, they miss out on parental care. It is known that the attention a carer gives tends to be dictated by how biologically connected the child is to the carer<sup>[18, 136]</sup>. This has implications even for non-orphaned children who do not live with their parents and worse still, for orphaned children. Early work on children's living arrangements was mainly based on cross-sectional studies that were not able to control for the timing of parental or other adult death in the household. It was shown, however, in several sub-Saharan African countries that a higher percentage of paternal orphans live with their mothers, who may assume the role of household head, than maternal orphans live with their fathers<sup>[18]</sup>. In 1998, it was estimated that, in Kenya, about 85% of paternal orphans lived with their mothers compared to only 54% of maternal orphans who lived with their fathers. In cases where the surviving parent migrates for work or remarries, the orphans are likely to live with their grandmother or other relative in just the same way the double orphans tend to<sup>[137]</sup>. It has also been shown that households with orphans tend to be larger and headed by females and older persons compared with those with no orphans<sup>[25, 138]</sup>. Although it is often believed that child-headed households are common, the evidence from Sub-Saharan Africa shows that these remain uncommon in most communities<sup>[16, 25, 139]</sup>.

More recently, studies using longitudinal data have become more available. These data reveal marked differences in the living arrangements for orphaned and non-orphaned children between countries, just like the cross-sectional studies. For example, in rural South

Africa the proportion of non-orphaned children living with their fathers is 38% compared to 66 % in Tanzania and 91% in Malawi and a very small percentage of maternal orphans live with their fathers, compared with the situation in Tanzania and Malawi <sup>[16]</sup>. Thus, even before the prevalence of orphanhood is considered, significant differences in child care practices exist in different populations.

The role of the extended family, particularly grandmothers, as guardians to their orphaned grandchildren is well documented <sup>[140, 141, 142, 143, 144]</sup>. In the traditional child-rearing system, the majority of orphaned children were taken in by their paternal or maternal uncles and aunts or surviving parent <sup>[16, 19, 145, 146]</sup>. Since traditionally most orphaned children are taken in by close relatives, in circumstances where an increasing number of orphaned children are being taken in by non-relatives, this might indicate a failing safety net <sup>[146, 147]</sup>. There is indeed fear that the extended family structure used in most of sub-Saharan Africa to cope with orphanhood have been stretched to the limit and can no longer to be adequately coping with the increasing number of orphans <sup>[19]</sup>.

In the Kenyan context, changes in household structures and children's living arrangements are happening at a time when there is a growing urban poor population who have limited access to secure livelihoods, or to amenities such as health care, schools, and housing among others. In a country where the majority of old people do not have formal social security and depend on informal employment, older people in urban areas are unlikely to be in a position to adequately fend for orphans. Households are generally very small and some residents have a loose or no connection to their rural kin. Given these circumstances, it is not known how living arrangements of children in this population change following death of an adult, especially a parent. Additionally, because the slums are ethnically and culturally very diverse with loose community identity and ties, they are thought to have low social capital on which to draw for support in times of need. As such, death of a guardian is likely to have a relatively large negative impact on children <sup>[148, 149]</sup>.

### **Consequences of Changes in Living Arrangements**

Documenting children's living arrangements is important because they may have implications for child care, nutrition, health and social development including education. Changes in children's living arrangements may result in children being moved to live with relatives or non-relatives with whom they are not familiar. In the process, household headship and therefore the control, prioritisation and allocation of household resources may change unfavourably for the child. For example, in a typical African setting, men dominate the means of production and fathers are often the household heads and the main income earners for households. Therefore, in circumstances where a woman or child takes over household headship, the household is likely to be financially disadvantaged. Also, while multi-generation households have been common in Africa, a rather new phenomenon of skip generation households has come onto the horizon. Grandparents have taken up child-rearing responsibilities after death of their own children <sup>[34]</sup>. In total, with such changes in living rearrangements, the dependency ratio in affected households becomes high, with fewer working adults looking after many children and, as a consequence, children's health, education and wider welfare gets compromised <sup>[147]</sup>.

Not living with parents does not, however, always imply worse health and social outcomes for children. Some children who do not live with their parents, including orphans, may live in wealthier households with better access to education and health care <sup>[130, 147, 150]</sup>. Non-orphans and single orphans may also get remittances from their non-resident parents. This might explain some findings that have shown that orphaned or fostered children do not have worse social and health outcomes compared with non-orphaned children.

These observations mean that understanding of local contexts is very important in elucidating the impact of adult death on children's living arrangements. No study has investigated children's living arrangement in relation to an adult death in households in an informal urban setting. Moreover, most of the available literature is based on research conducted by means of cross-sectional surveys <sup>[19, 25]</sup>. Using longitudinal data allows for understanding of the temporal ordering of events and thus may support causal inferences.

### **2.3.3 Impact of adult deaths: Child survival**

This section reviews the literature on the impact of adult death on child survival in the region and in Kenya. However, the first sub-section is dedicated to understanding the prevailing under-five mortality levels, causes and trends. This helps put in perspective the challenge of under-five mortality in region and how various factors including adult death may be contributing to the observed situation.

#### **Under-five mortality**

Globally, more than 10 million children under the age of five years die each year mainly from preventable causes and majority of these are from sub-Saharan Africa <sup>[151]</sup>. The target set by United Nations members at the General Assembly's 55<sup>th</sup> session to reduce child mortality by two thirds of the 1990 levels by 2015 <sup>[152]</sup> is out of sight for many countries. While clear progress has been made in reducing under-five mortality in sub-Saharan Africa, the 2012 progress review shows that the target will not be met in this region if current trends persist <sup>[153]</sup>. The slowest progress has been in reducing the neonatal mortality rate, which contributes about 40% to under-five mortality <sup>[154]</sup>. There are large differentials in childhood mortality by rural-urban residence, income and maternal education <sup>[155]</sup>. In 2003 the under-five mortality rate was estimated at 115 and 95 deaths per 1000 live births for Kenya and Nairobi province respectively <sup>[156]</sup>. The most recent estimates show a decline to 74 in Kenya as whole and to 64 deaths per 1000 live births in Nairobi province <sup>[13]</sup>. As is the case with the rest of sub-Saharan Africa, there are mortality differentials by place of residence, maternal education and wealth status. Estimates from Korogocho and Viwandani slums show that under-five mortality decreased between 2003 and 2009 from 113 to 79 deaths per 1000 live births <sup>[133]</sup>.

#### **Causes of under-five death**

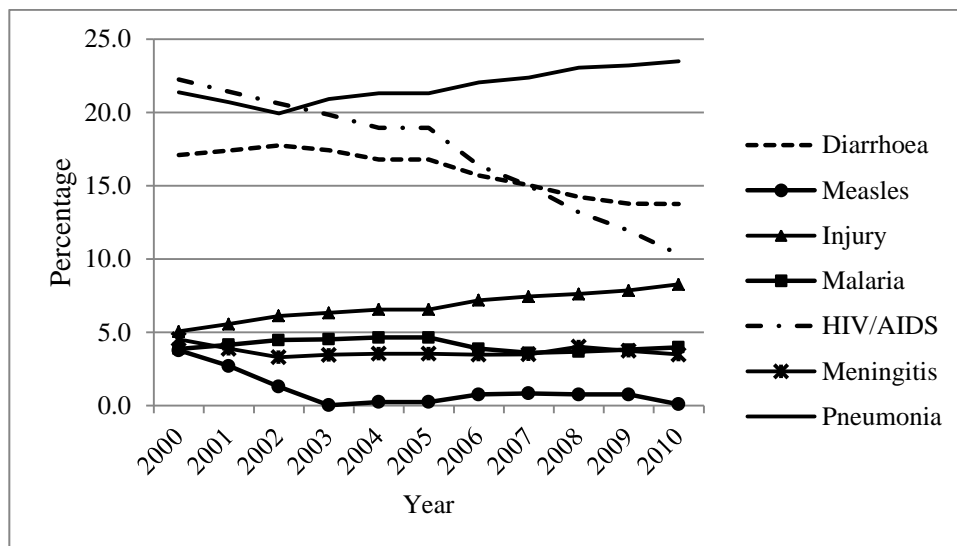
A shortlist of the most important causes of child deaths has been identified. However, there are variations in the level of burden from given causes globally, regionally, nationally and sub-nationally. Knowledge of these is important for planning, setting priorities and implementation of interventions. As for adult causes of death, data on causes of child death

are not readily available in most parts of sub-Saharan Africa. In a global study in 2012 by Liu et al., only 2.7% of under-five deaths had medically-certified causes <sup>[157]</sup>. This makes the use of alternative data sources such as verbal autopsies inevitable. Most estimates of causes of deaths are based on statistical models using multiple small published studies with child cause-of-death data. While there are uncertainties around the estimates and variations in the distribution of the major causes of death in different parts of the world, consistently the same major causes of child death have been identified <sup>[157, 158]</sup>.

Globally the leading causes of under-five death over the last decade include: pneumonia, diarrhoea, measles, malaria, injury, HIV/AIDS, neonatal causes (such as sepsis and birth asphyxia) and under-nutrition <sup>[157, 159, 160, 161]</sup>. The more recent estimates from 2010 show that globally, infectious diseases accounted for 64% of all under-five deaths while about 40% all under-five death were due to neonatal causes such as prematurity, birth asphyxia, sepsis, and meningitis. Pneumonia, diarrhoea and malaria were the main causes of death in older children <sup>[157]</sup>.

In Kenya the leading causes of under-five mortality include: pneumonia, diarrhoea, HIV/AIDS, injury, malaria, meningitis, measles and neonatal causes. Neonatal causes account for between 31-35% between 2000 and 2010. Over the last 10 years there has been a decrease in deaths from diarrhoea, HIV/AIDS and measles, but an increase in mortality attributable to pneumonia, Figure 1 <sup>[157]</sup>. For neonates, there has been very little change over the last 10 years in the relative magnitude of each of the major contributors to neonatal deaths. The most important causes of neonatal death include; prematurity, intra-partum related events and sepsis/meningitis/tetanus. Data used to derive these estimates used multinomial logistic regression on verbal autopsy data for high-mortality countries with no vital statistics <sup>[157]</sup>.

Figure 1: Trends for causes of deaths for children 1-59 months in Kenya, 2000-10



Source: Based on published Statistics from: Child Health Epidemiology Reference Group: [http://cherg.org/projects/underlying\\_causes.html](http://cherg.org/projects/underlying_causes.html)

### Pathways to child mortality

The pathways to under-five death after an adult death are complex but can be analysed using the proximate and social determinants of child survival as envisaged in the analytical framework proposed by Mosley et al. <sup>[162]</sup>. The framework recognises that distal social determinants at individual, household and community levels work through more proximate factors including; maternal, environmental, personal disease prevention and treatment; injury and nutritional factors to result into ill-health and death. Several explanations may be advanced to explain the current levels and slow decline in child mortality in sub-Saharan Africa. While a number of cost-effective child health interventions such as immunisation and treatment of diarrhoea with oral rehydration solution are available, coverage and uptake of these varies and remains low in some settings <sup>[163]</sup>. Many children die in the first one month of their life and most of the causes are associated with poor obstetric care. Less than 50% of pregnant women in Kenya deliver with help of trained assistants and untrained assistants lack skills in neonatal resuscitation and care and this contributes a big percentage to neonatal deaths. Access and uptake of preventive and curative services vary. Measles immunisation coverage for Kenya in 2010 was 85%. About 50% of children with

symptoms of acute respiratory tract infection were given an antibiotic while 78% of children with diarrhoea received oral rehydration therapy or increased fluids <sup>[13]</sup>. Part of the reason for the poor utilisation of health care interventions from the supply side lies in the limited health care manpower, supplies, motivation and infrastructure <sup>[164]</sup>. From the demand side, guardians may not seek health care for their children due to lack of appreciation of the need to do so, financial constraints, and lack of knowledge of where to find a particular service.

Over the last two decades, HIV/AIDS has increasingly contributed to poor child health. Evidence has emerged over time to show that children of HIV infected parents are at a higher risk of death than those of non-infected parents <sup>[6, 90]</sup>. Paediatric HIV infections <sup>[20, 165, 166]</sup> and probably the indirect effects of orphanhood (especially maternal orphanhood) are responsible for this trend <sup>[167, 168]</sup>. Studies have shown that in populations where HIV prevalence is high ( $\geq 5\%$ ), HIV has significantly contributed to an increase in or stagnation in the decline of under-five mortality <sup>[169]</sup>. Even with increasing availability of effective prevention of mother-to-child transmission of HIV, many mothers do not know their HIV status and are never tested during pregnancy and child birth. Therefore, children born to HIV positive mothers are likely to acquire HIV in utero, at birth or through breast milk and yet this risk could be significantly reduced using available and cost-effective interventions <sup>[170, 171]</sup>.

Malnutrition remains prevalent, especially among the poor. It is estimated that over 50% of under-five deaths have malnutrition as an underlying cause <sup>[159]</sup>. Infants who are not breast fed due to death of their mother or for other reasons are more likely to die than those who are breast fed. Under-nutrition undermines the child's immune system rendering children vulnerable to infections, particularly pneumonia and measles, but the reverse is also true, poor child health can lead to malnutrition <sup>[172]</sup>. Unfortunately, where only one cause-of-death is reported, under-nutrition tends not to be captured. Instead, the more acute causes such as pneumonia, malaria and diarrhoea, which tend to co-morbidities with under-nutrition, are easily picked up and reported and thus effectively understating its importance.

### **Impact of adult death on child mortality**

Understanding the relationship between adult death and child death is important because death of an adult may act through more proximate determinants of child survival to influence child health outcomes. The literature review in this subsection examines and synthesises findings from studies on the impact on adult death on child health. The review covers any adult death, whether parental or non-parental, and any cause of adult death. In the literature, the lower cut-off age for defining an adult has varied from 15 to 18 and therefore I examined all studies with age cut off of 15 years and above. The review also covers all-cause and cause-specific mortality literature.

A number of studies have found that death of an adult in a household increases the risk of child death <sup>[173, 174, 175]</sup>. More often than not, however, this depends on how the child is related to the deceased adult. It has been observed in several studies and settings that death of a mother, but not that of a father, significantly increased the risk of child death among children less than 10 years of age <sup>[6, 168, 173]</sup>. Death of a mother may mean loss of nutrition, particularly where households are too poor to afford alternative feeding. The authors argue that abrupt cessation of breast feeding is crucial in mediating the increased the risk of child death among children less than 2 years of age who have lost a mother. Evidence from other studies has shown that children who do not breast feed as a result of maternal death are more likely to be malnourished or die than non-orphaned children <sup>[168, 176, 177, 178]</sup>. Aside from loss of nutrition, loss of mother might also mean loss of care to the child including health care-seeking. Non-parental care givers might have limited skills and resources for ensuring that a child is protected against disease and injury or adequately treated when they fall sick, hence increasing their risk of death. Ronsmans et al. in 2010 demonstrated that the effects of maternal orphanhood persisted up to 10 years which points to the crucial role of maternal care beyond nutrition <sup>[173]</sup>. Contrary to this observation, a study in rural Malawi found that, while death of a mother increased the risk of child death, this depended on the cause of her death. HIV/AIDS-related maternal deaths were associated with higher risk of child death while non-HIV/AIDS deaths and paternal deaths were not associated with an increased risk of child death <sup>[6]</sup>. In a similar study in Uganda, both maternal survival and HIV status were found to be independent predictors of child survival <sup>[165]</sup>. These



observations indicate that, while maternal death increases the risk of child death, maternal HIV status in its own right is also related to higher child mortality. The additional risk seems to be due to vertical paediatric HIV infections from infected mothers. Prevention of mother-to-child transmission of HIV uptake remains low in many parts of sub-Saharan Africa <sup>[179]</sup>.

Most studies that examined the relationship between adult mortality and child survival, have mainly focused on parental death, yet death of other adult carers might have some impact on child well-being, particularly if they substantially contribute to household income or play a key role in child care. There is very little research focusing on assessing the impact of non-parental adult death on children in the same households. With high levels of orphanhood in the region and in Kenya, a substantial number of children (orphans) do not live with their parents either because the surviving parent is unable to look after the children or the child is a double orphan.

### **2.3.4 Impact of adult death: Child immunisation**

#### **Immunisation coverage and timeliness:**

Often, assessment of the performance of national immunisation programmes rely on immunisation coverage for the age cohort 12-23 months of age and data on coverage is readily available. However, not much detail is provided on the timeliness of when vaccines are given judged against recommended ages for each of them <sup>[12, 13]</sup>. Even when further analyses are done on survey data to assess timeliness, it is not possible to assess immunisation status and timeliness of children who die before the survey and yet such children are likely to have different levels of immunisation <sup>[180, 181]</sup>. Where both coverage and timeliness are assessed, it has been noticed that even when coverage is high, the proportion of those immunised within the recommended period is small <sup>[182, 183, 184]</sup>. One of the challenges in evaluating timeliness is that a large proportion of children, do not have immunisation cards from which accurate dates of immunisation can be retrieved <sup>[12, 13]</sup>. There is generally scanty research on immunisation timeliness and yet if vaccines are given too early or too late, a certain proportion of individuals are bound to remain at risk of

infection because they are under or not protected at all against infection <sup>[185]</sup>.

Immunologically, if vaccines are given too early they may not produce good enough immune response and may interfere with maternal antibodies while vaccines given later than recommended may produce better immune response but this exposes children to an increased risk of infection during the time they are not vaccinated.

In the slum settings, limited immunisation coverage may be a challenge but timeliness is likely to be a bigger one. Delay in immunisation is known to greatly vary between and within countries <sup>[180, 186]</sup>, therefore, there is need to assess its levels and determinants to help inform programme improvement in this population. Recommended ages for immunisation for the various vaccines were arrived at with due consideration of vaccine safety for the age, ability to induce desired immunity, timely protection of children against infections and logistical demand on the health care system. This therefore means that significant deviations from the recommendation may compromise and defeat the overall objective of immunisation. What constitutes delay differs by the disease being vaccinated against, but also by the population under consideration. How much delay is of public health importance remains unclear and has been defined differently in various research reports <sup>[180, 187, 188]</sup>. Partly, this is because different populations may have different levels of infection transmission risk depending on the proportion of susceptible individuals, closeness of contacts, and general health status of children including their nutrition <sup>[185, 189, 190]</sup>. For example, does a delay of one month in receiving measles vaccine in a rural population have the same significance as that in a congested urban informal settlement? This is an area that requires more investigation.

In spite of all the demonstrable benefits of child immunisation, coverage for certain vaccines remains suboptimal in many countries in Africa <sup>[191, 192, 193]</sup>. The Kenya Demographic and Healthy Survey (KDHS) of 2003 estimated immunisation coverage among children aged 12-23 months to be 57% and that for urban areas 59%, in the KDHS 2008/9, the immunisation coverage was estimated at 77.4% <sup>[13, 156]</sup>. Measles coverage in 2008/9 was estimated at 85% with differentials by maternal education, place of residence, province and wealth status <sup>[13]</sup>.

Barriers to immunisation may be grouped into health system factors, child/care taker and household factors. Delivery of vaccines from the manufacturer to consumer is a complicated process requiring maintenance of temperatures within a narrow range. To meet this, countries have in place elaborate vaccine storage and delivery mechanisms. In Kenya, vaccines are provided free of charge to all children who are eligible and health care workers are normally trained in the minimum requisite skills to administer vaccines safely. However in spite of these arrangements, there are healthcare system gaps and barriers that result in many children not getting immunised <sup>[194, 195, 196]</sup>. Some of the barriers identified in the literature include: Stock outage of vaccines and other supplies like needles, few health care facilities covering wide geographical areas, limited training and skills of health care workers and health care workers' attitude towards service users <sup>[197, 198, 199, 200]</sup>. At the household and individual child level, a number of factors influencing child immunisation have been identified including parental/ carer's limited knowledge of the benefits of immunisation (information), parental education, household wealth status and misconceptions about the effects of vaccines <sup>[191, 192, 198, 201, 202, 203]</sup>.

### **Impact of adult death on child immunisation**

The literature linking adult death and child immunisation status is very scanty. The available direct evidence base for a causal relationship between adult death and child immunisation is very weak or mixed at best. There are however, some indications that poor parental health and eventual death may be associated with low health-care seeking and immunisation of affected children due to diminished child care and finances at the household level <sup>[204, 205]</sup>. In a 2008 study, Mishra et al. estimated that in Malawi 43.8% of children living in households with a recent adult death or chronically ill adult were fully vaccinated compared to 65.2% in households with both parents. In the same study, a similar pattern was observed for Zimbabwe, with 48.8% fully immunised in affected households, compared to 55.4% in non-affected households <sup>[206]</sup>. Effects of adult death on child immunisation have not been explored in the population of the Nairobi slums.

### **2.3.5 Impact of adult mortality: Child schooling**

The Kenyan government has had several attempts at promoting universal primary education, and indeed the literacy level in Kenya is among the highest in the region. The current primary education policy aims at removing financial barriers to accessing education with an envisaged benefit of having all children at least completing primary education. The most recent effort aimed at ensuring universal primary education came in 2003. Kenya also signed up to the Millennium Development goals and MDG number 2 to “achieve universal primary education” is a key development objective. The target for this goal is: Ensure that, by 2015, children everywhere, boys and girls alike, will be able to complete a full course of primary schooling <sup>[152]</sup>. According the MDG progress report 2012, sub-Saharan Africa region accounts for over 50% of children out-of-school. However, there has been significant increase in net enrolment rates in the region from 58% in 1999 to 68% in 2004 and 76% in 2010 <sup>[155]</sup>. The report also notes that there are differentials in enrolment by gender, place of residence and poverty levels. Children from rural areas, slums and those from the poor backgrounds have lower enrolment and completion rates <sup>[155]</sup>. In Kenya, progress towards achieving this MDG seems to be on course. Net enrolment rate increased from 77.3% in 2002 to 92.9% in 2009; the gross enrolment rate increased from 73.7% in 2002 to 110% in 2009; the primary school completion rate increased from 62.8% to 83.2%; and there is near gender parity of enrolment of about 0.958 as reported in 2009 <sup>[207]</sup>.

### **Determinants of educational outcomes**

The free primary education policy introduced in Kenya in 2003 has been viewed as the vehicle to achieving the targets for the goal on education and indeed recent progress is widely attributed to this policy. However, while the overall picture looks promising, there are challenges in ensuring that all children complete primary education irrespective of their background and social circumstances. Research in Kenya has shown that school enrolment initially increased drastically but this has not been sustained over the years <sup>[208]</sup>. While government has been expanding the infrastructure, personnel and funding for free primary education, these have not met the huge demand to the extent that many commentators question the quality of education in public schools. As a consequence there has been an

increase in fee-paying private schools, some of them informal; to cater for the unsatisfied demand in underserved areas <sup>[209, 210]</sup>. Some authors have labelled these “private schools for the poor” given their location, for example in slums, and poor infrastructure and personnel <sup>[211]</sup>.

In evaluating the free primary education undertaking, most research has focused on how supportive or unsupportive is the school environment including availability of teachers, quality of education, and classrooms. In other words, attention has focussed on education system factors. Little attention has been paid to how children and their respective households’ circumstances affect educational attainment. Other than tuition fees, and physical infrastructure, several other inputs are needed to facilitate schooling including food, scholastic materials, clothing, housing and parental guidance <sup>[212]</sup>. This implies that, even if the educational system is improved optimally, children from certain backgrounds – with particular family, parental and individual child characteristics - might still miss out on schooling for various reasons.

### **The education decision-making theory**

Household-level determinants of school participation have been theorised and categorised into three dimensions<sup>[213]</sup>:

- i) Economic-perceived future return on investment: parents and guardians invest in their children expecting that they will look after them in old age <sup>[213]</sup>. That investment is likely to be focused on those children with greatest academic potential or those likely to be employed. For example, men have better employment chances than women. Parents may also only invest in their own biological children as opposed to fostered children <sup>[18]</sup>. Other studies have found a contradictory relationship. In a 2004 study, Akresh et al. found that fostered children in Burkina Faso were as likely to be enrolled in school as non-fostered children <sup>[214]</sup>.
- ii) Resource constraints: Resources are always limited. Therefore, given household income, its membership and their needs, resources will be directed to more immediate needs as opposed to investment in education, which has only

long-term returns. This potentially can influence educational decisions, for example, limiting number of years spent in school, and the type of school a child is enrolled in among other decisions. It follows that children from poorer backgrounds are more likely to have poor education outcomes if education is given less priority <sup>[18]</sup>.

- iii) Cultural and traditional norms: Cultural and religious norms may dictate child education. For example girls may be valued less as they tend to marry and leave family, or may be assumed not to need as much education.

### **Impact of adult death: Educational attainment**

How does adult death fit into the theoretical framework outlined above? Death of an adult, particularly a parent, might mean loss of income needed to facilitate child schooling. It may also mean loss of the care and support needed to keep a child in school. Guardians may not have the means or interest in investing in children who are not their own and are unlikely to contribute to their wellbeing in old age.

The literature on the impact of orphanhood on schooling is mixed. While a majority of the literature shows that being orphaned has a negative impact on children's educational attainment, some studies have shown little or no impact. Studies have shown that orphans, particularly maternal and double orphans, are less likely to be enrolled in school <sup>[5, 18, 215]</sup>, have slower progress <sup>[216]</sup>, complete fewer years of schooling <sup>[22]</sup> and are also more likely to drop out of school than non-orphans <sup>[23]</sup>. In another study in KwaZulu-Natal, South Africa, Case et al. found that less money was being spent on maternal orphans' education than non-orphans <sup>[22]</sup>. In a 2005 study by Ainsworth et al. in Tanzania, it was found that children spent less time in school prior to an adult death and girls spent significantly less time in school immediately after an adult death in a household <sup>[5]</sup>.

Orphanhood may be correlated with poverty and often studies do not adequately control for it and yet this might confound the relationship between orphanhood and educational outcomes. In a 2010 study, Ardington et al. noted that paternal orphans in South Africa were more likely to live in poorer households than non-orphans while there was no

difference between maternal orphans and non-orphans <sup>[217]</sup>. Other studies have shown that negative impacts are limited to children from households that were already poor prior to adult death <sup>[26, 216]</sup>.

The literature also shows that the impact of adult death on children's schooling varies between regions, countries and within countries. Bicego et al. in 2003 reported that the effects of orphanhood on attainment were more marked in East and Southern Africa than West Africa, probably reflecting the extent of HIV/AIDS related orphanhood in these countries, emphasising the need for context-specific evidence <sup>[216]</sup>.

With few exceptions, the current evidence is based on cross-sectional studies which do not take temporal ordering of events into consideration <sup>[18]</sup>. For example HIV/AIDS deaths come after a protracted period of illness, meaning that the effects may come before death. Also, many of the studies have focused only on parental death, although death of a guardian might equally be important. Other studies have further limited their scope to a specific cause of adult death, mainly HIV/AIDS <sup>[18, 215, 216]</sup>. The impact of adult death on child schooling in urban informal settlements in Kenya is yet to be examined. The resilience and coping mechanisms in the slums might be different from that observed in rural areas and other places owing to structural differences in households in terms of size, membership and income.

### **2.3.6 Summary**

The foregoing review provides the current knowledge, identifies gaps and highlights areas for potential research. There is a dearth of appropriate and empirical data on adult mortality in urban Kenya and as such there is limited evidence on the link between adult death and child social and health outcomes in general. Adult mortality in sub-Saharan Africa is high, mainly attributable to HIV/AIDS. However, data on adult deaths and specific causes of death are scarce, with very little data on urban populations. Impact of adult death on children is multidimensional, affecting children's social and health outcomes. The impact

also varies from place to place and appears to depend on pre-existing household circumstances and their response and less on children's characteristics.

The literature suggests that following an adult death, especially of a parent, the likelihood of migration from cities to rural areas increases. However, this relationship has been observed mainly in the context of HIV/AIDS before ARV became readily available. This observation is yet to be demonstrated in the context of better survival through the use of ARV. Children whose guardians die are predominantly taken in by relatives or surviving parent, especially the mother. This arrangement taps into the extensive extended family system common in sub-Saharan Africa, but the impact on children's living arrangements in slums following an adult death is not known. Generally, the literature shows that maternal death increases the risk of death for children under the age of 5 years. The literature is, however, mixed on the impact of paternal and non-parental adult death. Also, the impact of parental death on child schooling tends to be negative, but seems partly to depend on the outcome measured. For example, with regard to education, it appears that where enrolment is high, effects on enrolment may not be discernible yet effects on progression might be more demonstrable.

## **2.4 Conceptual Framework**

### **Introduction**

The impact of an adult death may be wide and far reaching, affecting individuals, families and the community at large. This thesis, however, limits itself to assessing the impact on child well-being. The usage of the term "child well-being" in the literature varies widely but it is generally taken to be a composite measure that encompasses various aspects of children's lives including health and safety, housing, educational attainments, economic prosperity, subjective well-being, peer and family relations, emotional and psychological states, behaviours and risks among others <sup>[218, 219, 220, 221]</sup>. The domains and indicators normally used to measure wellbeing tend to reflect the provisions and principles of the Convention on the Rights of the Child summarised thus: *the right to survival; to develop to*



*the fullest; to protection from harmful influences, abuse and exploitation; and to participate fully in family, cultural and social life* <sup>[222]</sup>.

The interest in examining impact of adult death on children is informed by the fact that children are among the most vulnerable members of society. While any child might lose any of their rights, children who lose their parents or guardians are at a heightened risk. Many studies have limited themselves to assessing impact on orphans, especially HIV/AIDS orphans; however this thesis looks at all children who are potentially vulnerable by virtue of losing an adult in their households. Here, I define vulnerability the way it has been used by UNICEF, UNAIDS and other organisations working in this area. Vulnerable children are those living in circumstances with higher risk of negative outcomes such as malnutrition, loss of education and morbidity- and whose prospects for continued growth and development are more threatened than their peers <sup>[223, 224, 225]</sup>. In common usage is the term “orphaned and other vulnerable children” which refers to children who have been orphaned and others rendered more vulnerable by other circumstances such as poverty, conflict, famine, and the effects of HIV/AIDS among others, who are more likely to fall through the cracks of regular interventions and programs than their peers <sup>[224, 226]</sup>. Other specialised agencies like The President’s Emergency Plan for AIDS Relief (PEPFAR) working on HIV/AIDS further focus on child vulnerability to the effects of HIV/AIDS and define a vulnerable child as one having either of the following: being HIV positive; living without adequate adult support-due to recent adult death, chronic illness, living with a grandparent or child as household head; living outside family care; and being stigmatized or discriminated against <sup>[225]</sup>.

By examining impact of adult death on children and going by the definitions given above, this thesis examines the extent of adult death impact on children and identifies aspects of their well-being that are affected. This study does not attempt to assess the impact of adult mortality on the whole spectrum of child well-being measures, but rather limits itself to common indicators used in the literature whose data are also available within the existing data source.

The conceptual framework is a representation of the relationships between the exposure, intermediate and outcome variables as conceptualised from the literature review and some of the overarching theoretical frameworks that have been applied elsewhere in similar research as discussed in the literature review. While determinants of child health and social well-being are numerous, this simplified representation, within the allowance of available data and study design, uses variables that are available in the database but also some of those variables that are potentially measureable, were this to be a study to be carried out in the future.

While the conceptual framework aims at summarising what has been identified from the literature review and available theories, with regards to the relationship between adult death and child well-being, it is also designed to guide the analysis to be carried out. The model shows the relationships between variables and their direction, and whether they are likely effect the outcome directly or through an intermediary (causal pathway variables). Some of these are established relationships from the literature while others have not been explored or tested in the way presented here. The conceptual framework deliberately excludes aspects of the literature that relates to determinants of adult mortality but rather focuses on its consequences on child well-being. This was done on purpose to avoid taking away the focus from the main theme of the thesis-exploring the relationship between adult death and child well-being. However, it is important to note that, as part of background characterisation of adult mortality, analysis of socio-demographic factors associated with adult deaths was conducted to help put in context the nature of adult mortality in this population. Also, some of the relationships depicted in the conceptual model are not explored in the analysis due to lack of appropriate data.

Given that children are generally dependant on adults, reducing the pool of adults in the household through death may compromise child care, support and income generation for the household, at least in the immediate aftermath. To start with, and for the sake of argument, we can assume that after an adult death there is no adult replacement in the affected household. The dependency ratio increases with fewer adults supporting more children. If the deceased was the household head, the new head might have limited means

to support the household. Likewise, if the deceased adult had major child care roles, this may be compromised, unless the surviving household members step-up their responsibilities to fill the gap. On the flip side, as an adjustment strategy, some adults and or children in the affected households might move out to ease the pressure on the available resources. Children may be sent to live with other relatives who can provide child care, such as aunts and grandparents, hence shrinking the size of affected households.

Death of an adult may result in reduced income to the affected household due to reduced number of adults in gainful employment. Reduced income may impact food security as the limited resources are parcelled out to various priorities. Access to health care might also be limited by lack of finances. Similarly, education might be impacted as seen earlier <sup>[5, 21, 22]</sup>. While the Free Primary Education policy in Kenya means that children do not pay fees, other essentials for schooling require money, for example transport, scholastic materials and uniforms. Faced with limited resources and probably with less altruistic non-parent household heads, educational decisions made for the children might be unfavourable.

While it is intuitive that death of an adult in household leaves the household with one adult less, other adults might join the household to fill the void left by the deceased as a response or coping mechanism. This might occur, for example, through marriage, if the deceased was a spouse, or another adult might join to provide paid labour for household domestic chores. Alternatively, the replacement might be an unpaid relative such as a grandparent, as has been reported in many places mainly with regard to HIV/AIDS orphans <sup>[19, 217]</sup>. The extent to which a replacement adult helps mitigate and close the deficit caused by an adult death will depend on their contribution to income generation, child care and their ability to meet their own needs.

Furthermore, in response to bereavement, affected households might get financial or other support from relatives and friends to help them cope with the loss. Where governmental or non-governmental organisation support for orphans exists, they may be targeted and supported in various ways including schooling, nutritional or financial support. In the slums, a few support organisations work with orphans but their activities and program

impact cannot be easily quantified. It is also possible the bereaved household members might, out of sheer determination, work harder than usual to fill the gap in income, care or support that the deceased used to provide.

Other potential determinants and or confounders of child social and health outcomes may include individual child characteristics or biological predisposition such as birth weight, age and sex. Household factors may include socio-economic status, maternal education, parity and child's living arrangements. Community level and environmental factors may include sanitation, water, and disease vectors such as mosquitoes responsible for malaria transmission. Health care factors and social services factors include accessibility in terms of geographical distances, financial access, quality and responsiveness to client needs. This thesis focuses on household level and individual child factors with the key exposure of interest being adult death in a household. I hypothesise that the impact of an adult death on children is partly mediated through changes in child's living arrangements and their migration experiences. Changes in household head, for example, may lead to a household having an elderly or child head with limited means of livelihood. Changes in household size and household income may act through reduced child care, nutrition, and limited access to care to influence adverse child outcomes.

### Child health and social outcomes:

The scope of child health and social outcomes is large, including but not limited to morbidity, mortality, health care utilisation, nutrition and growth, behaviour and socialisation. Within the constraints of the study design, available data, finances and time, a limited number of health and social outcomes are studied here. Under child health outcomes, I looked at child survival and health care seeking in the form of child immunisation. Different aspects of immunisation were examined, including coverage and timeliness of receiving vaccines. Of the social outcomes, only child educational outcomes were assessed. Four aspects of child education were assessed, including enrolment, interruption, grade-for-age and grade repetition.

### Child survival:

Changes in a child's living arrangements might result in a child living with non-parents, poorer, less altruistic and less knowledgeable guardians, non-relatives, and probably in a household with high dependency ratio. These changes might directly affect child care, nutrition, use of preventive measures and health-care seeking and, in turn, these may have a direct impact on child health and survival <sup>[174]</sup>. Loss of income may also affect child care, nutrition, health care seeking and utilisation and survival. Child migration might mean a child going to more disease-endemic destinations, with poorer food security, unsafe water and limited access to health care facilities <sup>[122]</sup>. Unfortunately, unlike internal migration, the destination of out-migrating children is not known and neither are their morbidity and mortality outcomes. The standardisation of the pathway to child mortality partly borrows from the Mosley and Chen analytical framework for child survival although many of the proximate variables are not measured in this study <sup>[162]</sup>.

### Health care utilisation (Immunisation):

Death of a key child care giver may result into a child being cared for by a less knowledgeable person, who might be poorer and less altruistic. Child migration following death of a parent, for example, may result into children being sent to live with grandparents in rural areas with poorer access to health care services. Child dislocation might result in loss of health care documents, like the child immunisation card, and this might result in a child discontinuing and missing the remaining vaccines. Also, the new destinations might be geographically less well served with health care services for immunisation. Recent migrants may have difficulties in accessing services in their new location, probably due to limited knowledge, low social capital and limited financial means consequently impacting their well-being <sup>[113, 114, 122, 199]</sup>.

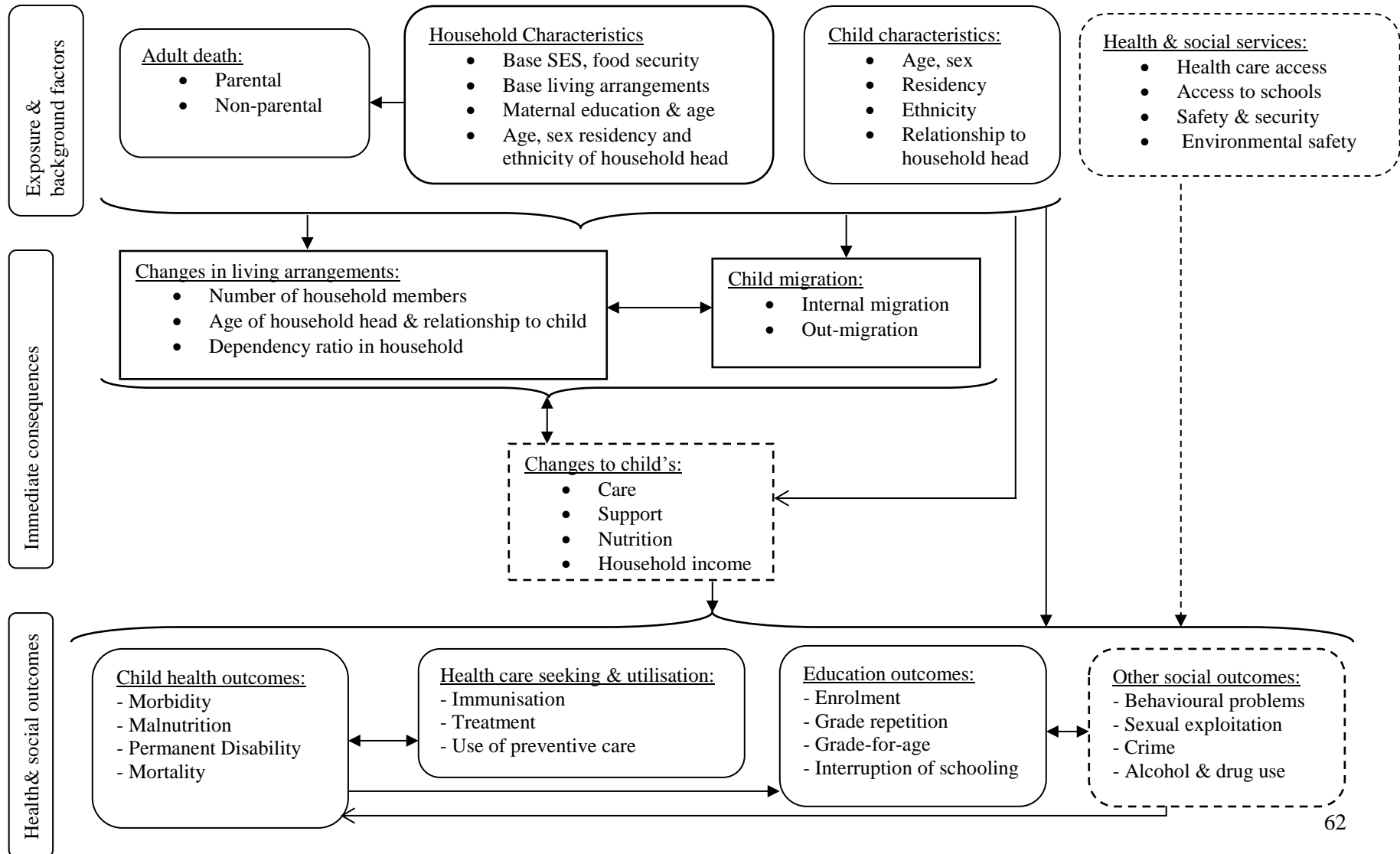
### Education:

Education decision-making theory posits that amidst scarce resources, choices about child education might be informed by: expected “returns on investment” meaning that non-parents, for example, may not prioritise investing in non-biological children. Also with

limited resources, choices might be made about which child goes to school and for how long. Lastly, cultural and religious norms might influence child placement in school. Guardians might not prioritise education, constrained by limited resources, less altruism, ignorance, or outright exploitation of children as domestic servants, resulting in poor education attainment <sup>[5, 18, 130, 213]</sup>. Child migration might mean a child going to a place poorly served by schools and this might result into dropping out of school. Migration might also interfere with the school placement resulting in interruption or eventual drop out of school. Emotional distress due to bereavement might affect school attendance and concentration in class and this might subsequently lead to poor education outcomes.

Figure 2 summarises the conceptualisation discussed above in a schematic diagram. Adult death is the key exposure that works through changes in children's living arrangements, changes in household socio-economic fortunes and migration to influence both health and social child outcomes. Single-headed arrows indicate that the relationship is unidirectional while double headed arrows indicate that the relations are bi-directional. Variables in dotted outlined box labelled "Health and Social services" are important in the conceptualisation but were not measured and thus not controlled for. Similarly, variables in the dotted box labelled "Diminished" refers to household resources such as wealth, care, emotional support, time, nutrition, knowledge and prioritisation of child needs. It is envisaged that with death of an adult in the households, changes to these are bound to happen and ultimately impact the well-being of children. These can be thought of as being in the causal pathway between the exposure and ultimate health and social outcomes. With the exception of wealth, these, like health care system factors, were also not measured due to limitations in the study design and quantity of data available.

Figure 2: Conceptual Framework



### 3. Data and Methods

This chapter describes the methods used in this research. First, I present a summary of the research setting and platform used to generate the data. Secondly, I highlight general aspects of the data preparation procedures and statistical analyses carried out. Specific statistical methods are summarised and discussed in the respective chapters before the presentation of results.

#### 3.1 Study setting

##### 3.1.1 Nairobi Urban Health and Demographic Surveillance System (NUHDSS):

This thesis uses data collected from an ongoing health and demographic surveillance in two informal settlements located on the outskirts of Nairobi city, Kenya. The Nairobi Urban Health and Demographic Surveillance System is run by the African Population and Health Research Center (APHRC). APHRC is an autonomous non-governmental organisation headquartered in Kenya and conducts research in several other African countries. APHRC is also a member of the umbrella organisation for all sites conducting health and demographic surveillance in developing countries, the INDEPTH network (*International Network for the Demographic Evaluation of Populations and Their Health*) <sup>[227]</sup>.

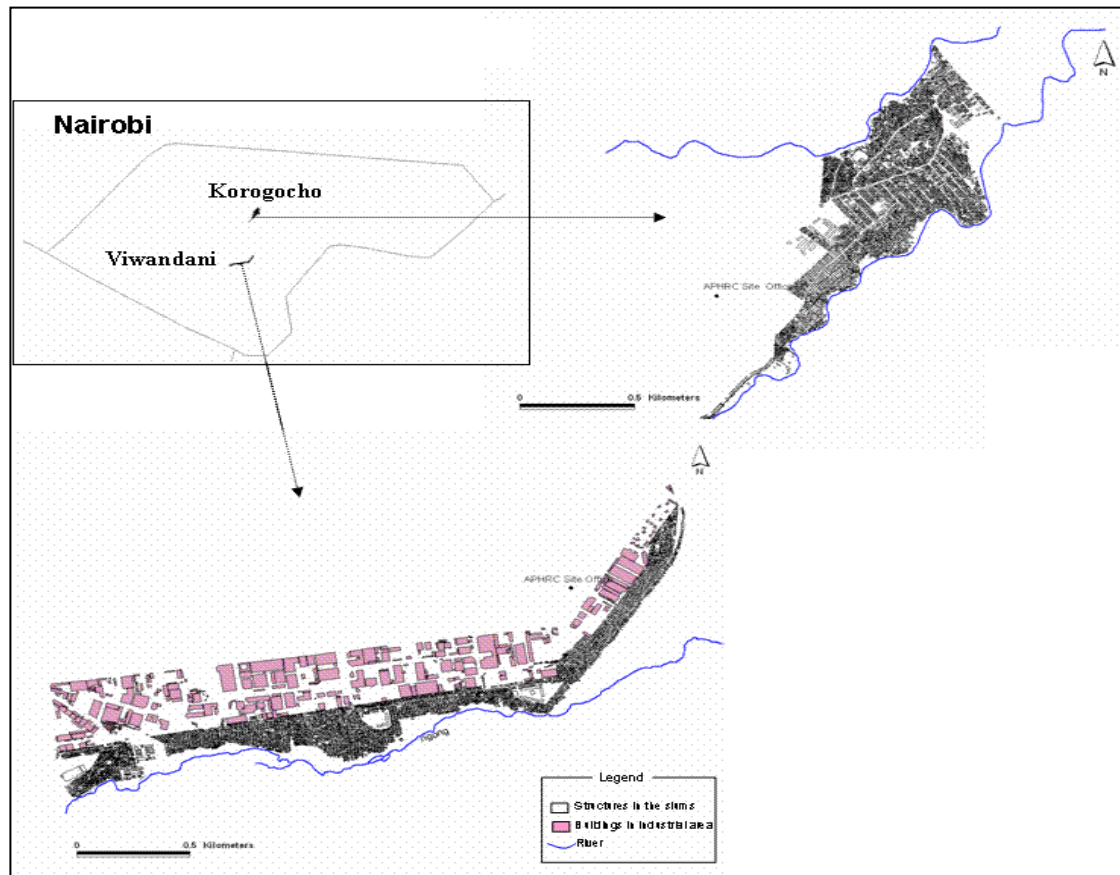
##### 3.1.2 Background

The Nairobi Urban Health and Demographic Surveillance System (NUHDSS) became operational in August 2002. It covers two geographically separate, informally built residential areas with a combined total area of about one square kilometre. The two areas, Viwandani and Korogocho, are located approximately 7 kilometres apart and are about 7 and 12 kilometres from Nairobi city centre respectively (Figure 3). It is estimated that informal settlers began building temporary and semi-permanent structures in these two areas back in the 1970s and these have been expanding both in geographical expanse and density. Both slums developed on public land near river banks. Korogocho is near the river bank of the Nairobi river while Viwandani is on the banks of the Ngong river (Figure 3) <sup>[228]</sup>. The Korogocho surveillance area is composed of seven villages (Korogocho A; Korogocho B; Highridge; Gitathuru C; Grogan A;



Grogan B and Nyayo), while Viwandani is composed of five villages (Kingston; Donholm; Jamaica; Lunga Lunga and Paradise) <sup>[229]</sup>.

Figure 3: Map showing Korogocho and Viwandani slums in Nairobi, Kenya



Source: (APHRC, 2009)

Residents of the informal settlements are mainly rural-urban migrants; some of them are long-term migrants, while others were born and raised in the slums. Migrants come from across the various ethnic backgrounds and the compositions of the two populations broadly reflect the relative sizes of the ethnicities at the national level. The population has high mobility, particularly in Viwandani. However, net migration is positive with slightly more in-migrations than out-migrations. Circular migration is high with about 20 to 30% of the entire population moving out and in a given year <sup>[100]</sup>. The total population under surveillance grew from about 60,000 in 2003 to 73,000 individuals as of end of 2008. Korogocho slum accounts for about 44% of the total population under surveillance.

The two informal settlements are characterised by temporary and semi-permanent housing structures, unpaved and narrow alleys, and poor sewage and water systems. Most water and power connections are illegal. Because of this, there are numerous broken water pipes with a potential for contaminating tap water while the illegal power connections increase the risk of fires and electrocution accidents. About 40% of households have electricity. Solid human waste is mainly disposed of in the rivers, rubbish tips or pit latrines. There are also a few community water-borne toilets.

While the two informal settlements are similar in many respects, some striking differences also exist between them in terms of social setup, livelihoods and demographics. Korogocho slum is largely composed of relatively settled migrants who are mainly unskilled labourers and petty traders. The average household size for Korogocho in 2008 was 2.9 persons per household while that of Viwandani was 2.3. Korogocho slum has generally poorer health indicators than Viwandani. The total fertility rate for Korogocho in 2008 was 3.4, while that for Viwandani was 3.3, compared to 2.8 for Nairobi as a whole <sup>[133]</sup>. Korogocho slum settlement is located very close to the Nairobi city refuse dumping site and most housing structures are made of mud, timber, iron-sheet roofs. Residents are generally poorer and have lower educational attainments than their Viwandani counterparts. Viwandani slum is located near an industrial park, and residents are generally younger, more educated and predominantly male. More men in Viwandani slum (30%) are in salaried or stable income employment than in Korogocho (16%) <sup>[133]</sup>.

In 2008, it was estimated that Viwandani had a higher proportion of working age adults (72%) aged 15-64 years than Korogocho (62%). The reverse was true for children under 5 years of age, who made up 37% of the population in Korogocho and 28% in Viwandani in the same year. While both slums have proportionately fewer individuals below the age of 15 and aged 60 and above than that of Nairobi and Kenya as a whole, this is more pronounced in Viwandani slum (Figure 4).

Figure 4 shows the population pyramid for the two slums separately (Figure 4 a & b) while that for the two slums combined is in Appendix I.

Figure 4 (a): Population pyramid for Korogocho slum in Nairobi city

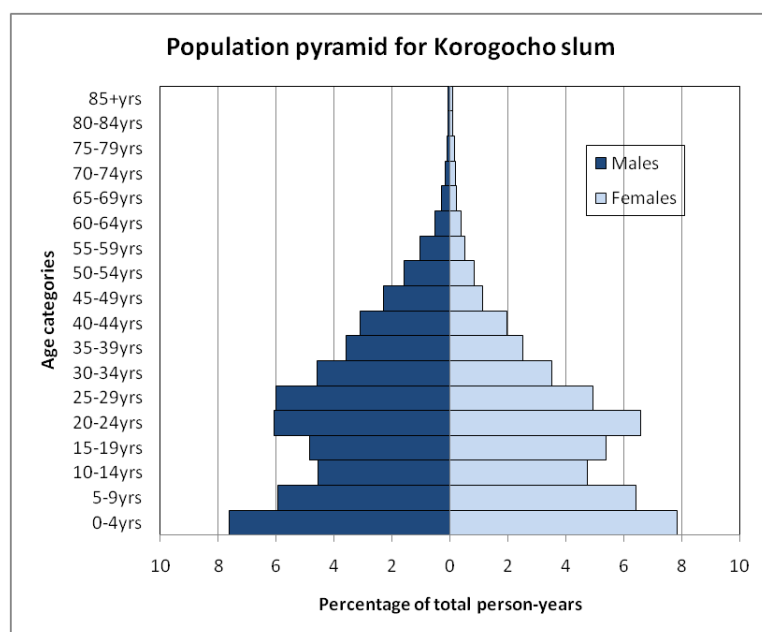
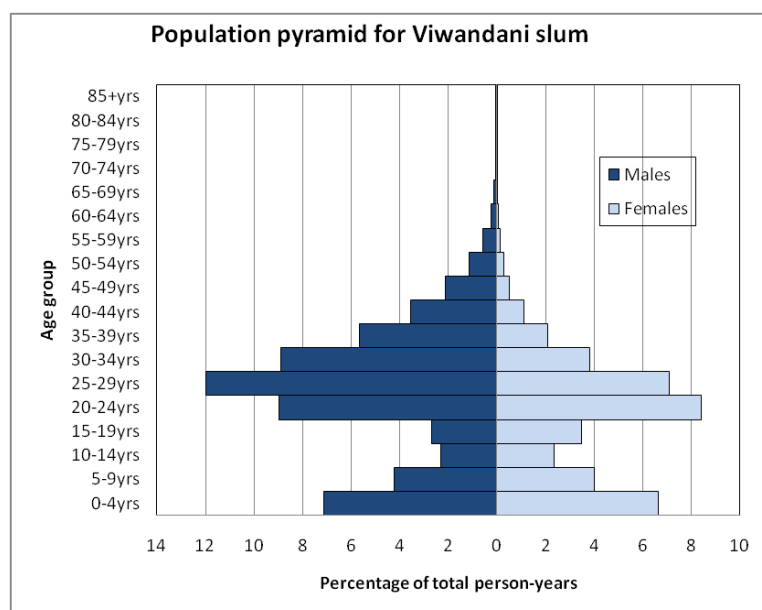


Figure 4 (b): Population pyramid for Viwandani slum in Nairobi city



### 3.1.3 NUHDSS operations and definitions

The initial census took place in August 2002 with registration and assigning of identification numbers to individuals and their dwelling units. All individuals who consented were enumerated and given unique identification numbers. Their dwelling units were also identification numbers. Other basic information on all members were collected, including education, marital status, ethnicity, age, education, parity and the immunisation status for children.

After the initial census, subsequent updates on statuses of current residents and recording of new members were carried out every three months. Later, for operational reasons, the updates and data collection waves were changed to every 4 months. Operationally, a household is defined as a social unit of people normally sharing meals who recognise one of the members as the household head. Household members are not necessarily bound by blood relations. Non-resident “members” are not considered as *de facto* household members as discussed later.

The NUHDSS data are managed using both paper-based and computer-based data management systems. Field work and data collection is primarily done using pen and paper. Individuals who were registered at the initial census were recorded in a master book, the Household Registration Book (HRB) and the computer database. At each of the data collection and individual status update rounds, trained field workers go to the field with the HRB and identify households contained in the listing in the HRB that they are carrying. After a household has been identified, the statuses of all previously registered members are confirmed by interviewing a suitable respondent in the household. Members who were previously in a given household may have left the household, new members could have joined it; others could have died; while still others might have been born in the interval since the last update.

An event form is completed for any changes in household membership and important events such as migration, births, deaths, immunisations, are captured and are submitted together with the HRB. For every household with a child aged less than 5 years, a complete review of their immunisation status is undertaken, while for households with children under the age of 18 years, their current schooling status is elicited and noted

down. Once every year, an assessment of household assets used to derive an index of household wealth is carried out.

Once the households and household member statuses have been updated in the HRB and all forms related to the events completed, the HRB is edited by the team supervisor. If there are no errors or omissions, the HRB is returned to the office by the overall field supervisor. Those with errors or omissions are returned to the field worker to make corrections. When the HRB gets to the office, the office editors crosscheck for potential errors and completeness, and check that all the expected forms exist as per listed events. The events forms are important as they are the ones used to effect changes in individuals' statuses in the computer database.

#### **3.1.4 Data management**

After the HRB and event forms have been received and edited at the office, they are passed on to the data entry manager. The data entry manager oversees a team of trained and experienced data entry clerks to whom he assigns the HRBs for data entry. All collected data are entered in a relational database maintained on the Microsoft SQL server platform. A team of data entry supervisors re-enter about 10% of the data to crosscheck for data entry errors. They also provide continuous on-job training to the data entry team. An overall database programmer, with support of other programmers, oversees and maintains the database. This team creates new data entry screens as a need for them arises. They also extract analytical data files as requested by the research team. After data entry for a particular data collection wave is completed, the database is updated and the new statuses and events captured are reflected in the system. New Household Registration Books are then printed in readiness for the subsequent data collection wave and the cycle is repeated.

#### **Household structures, relations and migration**

A single physical housing structure can be occupied by more than one household living in different rooms. All households are assigned a unique household number. New structures are assigned numbers once they are occupied. Every household has a household head. In the NUHDSS database, relationships within the household can only

be identified through the household head to whom everybody else in the household relates directly. If a household head moves with the entire household from current location to another, he maintains the household headship. However, if the household head moves and leaves the rest of the household members behind, one of the remaining members take on the headship role. If a former household head forms a new household alone, he automatically becomes the head. However, if he joins another existing household, his relationship with the existing head has to be defined and indeed he might take up headship role at the new household depending on how the household members regard his role in the household.

Members of the household who move out of the household and out of the slums for a period of more than 120 days are regarded as out-migrants. Their final status in the database is indicated as out-migration. If they return, they are in-migrated into the system. If a member leaves the household for another household within the slums and stays there for more than 120 days, that person is considered to have had an internal change of residence. Their status at original place of residence is indicated as “exited” and an “entry” is recorded at the new place. Individuals who move into the surveillance areas and stay for at least 120 days are registered as in-migrants. The choice of 120 days to determine migration status is rather arbitrary but the period conveniently maps on the data collection waves that are repeated every 4 months. When in-migrants become residents of the surveillance area for the first time, they are given a new unique identification number whereas, if they are returning out-migrants, their old identification number is searched for and re-instated. The new-born children of registered residents are also allocated new unique identification numbers. In general, an individual’s household number can change if they changed place of residence but their unique identification number never changes.

### **Deaths in the NUHDSS**

As mentioned earlier, death is one of the events captured in the status update rounds. As an important event, all deaths of registered members are identified and registered as far as possible. They may either be captured during the routine household status update round or reported by community informers. Like all other events, deaths are noted in the HRB and also captured on the death event form (death form) and returned to the office

for data entry. After a death has been identified, a specially-trained field supervisor visits the bereaved family and offers them condolences and prepares the ground for conducting a verbal autopsy interview with an appropriate respondent. The verbal autopsy interviewer is typically a Bachelors degree holder in the social sciences or a related field. After offering condolences, he makes an appointment with the member of the household who has the best knowledge of how the illness manifested, and the treatment received before the death. The verbal autopsy questionnaire used in the NUHDSS is based on a World Health Organisation designed verbal autopsy tool with a few local adaptations. There are two versions of the tool; one for under-five deaths and the other for individuals aged five years or more. They capture information on symptoms and signs experienced, duration of illness, health care sought and medical records, where available. While most of the questions are closed, the tools also have an open-ended history section where the interviewer jots down a narrative akin to a medical history taken by a clinician. After the verbal autopsy questionnaire has been completed, it is edited by the supervisor and returned to the office for data entry. Data entry supervisors are responsible for verbal autopsy data entry. This is done in a stand-alone database with identifiers removed, but is linkable to the main database through an ID. This is to maintain confidentiality as death information is quite sensitive, especially when it comes to highly-stigmatised causes of death such as HIV/AIDS.

To ascertain the cause of death, a team of three physicians are tasked to review and assign cause of death based on all the information captured on the verbal autopsy questionnaire. The physicians independently review and assign cause of death. For practical reasons, a shortened list of causes of death that can be mapped on the International Classification of Diseases tenth revision (ICD-10) of death was developed and is routinely used for coding causes of death. After the coding is done, the codes are compared and, if two or more physicians agree, the result is taken as the probable cause of death. Where agreement is not reached, the three physicians meet and discuss the case in order to reach a consensus. If no consensus is reached, the cause of death is recorded as indeterminate. To reduce the variability in the coding for cause of death, the turnover of physicians has been minimised to a total of five physicians over the period of five years with three physicians participating at a given point in time. The promising development of Bayesian probabilistic modelling of causes of death when eventually

embraced will go a long way in addressing the issue reproducibility and comparability of results across sites and time <sup>[80]</sup>.

## **Vaccination**

In the NUHDSS, information on child immunisation is routinely collected and updated every four months when field workers visit households. In the process of updating the residents' status, interviewers also inquire about immunisation status of children aged less than five years. Each child's immunisation data are updated until all the vaccines in the childhood immunisation schedule have been given to a particular child or the child reaches five years of age. The information on child immunisation status is elicited in two ways. Normally, for every immunised child, a vaccination card is issued. All vaccines given to a particular child are indicated on the card, including the date the vaccine was given. Where a vaccination card is available, immunisation status data are transferred from the child's immunisation card including dates. If no card is available, the child's mother or other responsible carer is asked to identify the various vaccines the child might have received. Descriptions of the various vaccines are given to aid recall and identification of the respective vaccines. Kenya adapted the WHO-recommended childhood immunisation schedule as follows: Bacillus Calmette-Guérin (BCG), a vaccine against tuberculosis, and oral polio vaccine, a vaccine against poliomyelitis, (oral polio vaccine-OPV-0) are given at birth or within 14 days after birth. Another three doses of OPV are given at 6 weeks, 10 weeks and 14 weeks. The first dose of diphtheria, pertussis, tetanus, hepatitis B & haemophilus influenzae type b (DPT/Hep-B/Hib) is given at 6 weeks after birth; the second at 10 weeks and third at 14 weeks. Measles vaccine is given at 9 months. Where there is suspected measles epidemic outbreak, measles vaccine can be given as early as 6 months based on the local public health officials' judgement. Outside the normal schedule, mass immunisation campaigns have also been carried for particular diseases, notably polio. This information is often noted on the immunisation card or other written material and, if it is not written down, information is elicited from the child's carer.

## **Schooling**

Initially, education attainment information was only asked of adults. In 2005, active prospective surveillance of schooling started with information being collected for



children aged 6 to 18 years. Schooling information for the years 2003 and 2004 was collected retrospectively. The schooling status and grade information has been updated annually since 2005. In the Kenyan educational system, children enrol in standard one at the age of six and, if there are no interruptions of their schooling or repetition of grades, they complete primary eight by age 14.

### **3.2 Data preparation**

Use of data collected by APHRC is authorised after the submission of a formal request accompanied by an abstract. At the time I applied for the data, the data available for use were for the years 2003 to 2007. After permission to access the data was granted, the data manager prepared the needed data files and forwarded the raw data to me in Stata format in separate files. The data files included; social group (household) file; individual file; verbal autopsy file; immunisation file; and schooling file. I merged and appended various files to each other, as necessary, to create usable analytical data files. A series of extra variables that were deemed necessary for the research were requested later and added to the analytical data files. I carried out consistency checks, cleaning, generation of new variables, recoding of values and restructuring the data into formats suitable for the various study objectives and subsequently carried out all the analyses. Prior to my PhD studies, I had been one of the physicians involved in the coding of causes of death, and training and supervision of the field workers.

### **3.3 Ethical issues**

The NUHDSS was granted ethical clearance by the Kenya Medical Research Institute scientific and ethics committee to conduct ongoing surveillance. Nested studies in the NUHDSS apply for specific ethical clearance. A copy of the ethics clearance form was submitted to the Ethics Committee, London School of Hygiene and Tropical Medicine.

Datasets for analysis are always stripped of all potentially identifying information before being handed over to the user. This is to ensure that confidentiality is maintained at all times. I signed an agreement committing myself to ethical and professional use of the data for purposes it was applied for.

## Dissemination

A bound copy of this thesis will be made available to the School library. Each of the results chapters will be turned into a manuscript for publication. Summary results will also be printed in APHRC's newsletters and fact-sheets.

### **3.4 Methods related to specific study aims:**

#### **Adult mortality levels, trend, causes and associated factors**

The methods presented in this section relate to the measurement of adult mortality. While measuring adult mortality is an objective on its own, it is also the main exposure variable for all the other child outcomes analysed in this thesis.

In this thesis, an adult death is defined as a death occurring to an individual aged 15 years or more. Conventionally, an adult is defined as an individual aged 18 years or more. Studies with varying aims and needs have used different age categories to measure adult health. In demography, adult mortality is conventionally computed for the age bracket 15 to 59 years. Also, while those aged less than 15 are considered minors, many studies of reproductive health and HIV consider the starting age of 15 since a sizeable fraction of under-18 years olds already face reproductive health challenges and are at risk of infection with HIV. The slum population is generally a young population with those aged less than 15 being over represented while those above 60 are quite a small fraction. For those reasons, I decided to define those aged 15 or more as adults and children as those under 15 years of age.

The study population was limited to those adults who had been present between 2003 and 2007 and registered by the NUHDSS. The point of entry into the study was marked by one of the following events: enumeration (at the start of the NUHDSS), in-migration or attainment of age 15 by individuals who had been resident before age 15. End of observation was marked either by death, outmigration, loss to follow-up, or the last date of observation- 31<sup>st</sup> December 2007, whichever came first. Between January 1<sup>st</sup> 2003 and December 31<sup>st</sup> 2007, a total of 86,070 individuals 15 years or more lived in the two slums at one time or another. Out of these, 1513 died while still resident in the slums. Person-time under observation was computed for all individuals.

Cause of death codes were anonymously linked to the individual and household level variables including age, sex, ethnicity, wealth status index, slum of residence, marital status and education. Using standard life table approaches, adult mortality was computed and summarised as  $_{45}q_{15}$  (the conditional probability of dying by age 60 given that one has lived up to 15 years of age). Estimates from this study are compared with those estimated by INDEPTH from 19 other demographic surveillance sites. Age and sex-specific mortality rates were also computed. Factors associated with all-cause and cause-specific adult mortality were assessed using Cox proportional hazard models and piecewise exponential models. Statistically significant interactions were added to the models.

### **Exposure variable**

In the subsequent analyses, the key exposure variable of interest for the various outcomes which include child migration, living arrangements, child mortality, immunisation and education was adult death in households where children lived. The variable was further categorised by the child's relationship to the deceased adult into: no adult death, father died, mother died, and other adult died. For some analyses, exposure was treated as binary: "no adult death" in the household and "adult death occurred" in household while, in other analyses, cause-of-death is categorised as: not exposed; HIV/AIDS/Tuberculosis; injuries and all "other" causes. Control variables generally include age, sex, slum of residence, the household wealth index, maternal education and ethnicity. The household wealth index was computed centrally by APHRC before the data were shared. The index is computed by principal components analysis of a set of household variables including household assets such as bicycle, television, radio, iron box, refrigerator; and housing materials. The index was categorised into 3 categories (tertiles).

### **Methods for migration and living arrangements**

For the analysis of child migration and living arrangements, the study population included children aged less than 15 years who were resident in the surveillance area between 2003 and 2007. Children came into observation through being born in the surveillance area, enumeration at the start of the study, or in-migration. End of observation was marked by out-migration, loss to follow-up or the end of observation

period- 31<sup>st</sup> December 2007, whichever came first. Two forms of migration were analysed; out-migration and internal change of residence. For internal migration, movement from a given household is marked by an “Exit event”, and joining a new household is marked by “Entry”. Data for migration and living arrangements were prepared per calendar year. Descriptive results are given by year and exposure status. I used logistic regression models for categorical outcomes and ordinary least squares regression for quantitative outcomes.

### **Methods for child mortality**

For child mortality, the study population consisted of all children under the age of five years who were resident in the surveillance area between 2003 and 2007 and registered by the Nairobi Urban Health and Demographic Surveillance System as members. The start of observation was marked by enumeration, birth, or in-migration. The end of observation was marked by death, outmigration, attainment of 5 years of age or end of observation period, i.e. 31<sup>st</sup> December 2007, whichever came first. The main outcome of interest was child survival time allowing for censoring. For the descriptive analysis, Kaplan Meier survival plots were estimated. For assessing risk factors for child mortality, multivariable analyses were carried out using Cox proportional hazard models and piecewise exponential models to estimate the hazard of mortality controlling for covariates.

### **Methods for child immunisation**

For child immunisation, the study population consisted of all children under the age of two years who were resident in the surveillance areas between 2003 and 2007 and registered by the Nairobi Urban Health and Demographic Surveillance System as members. The start of observation was marked by enumeration, birth, or in-migration. The end of observation was marked by death, outmigration, attainment of 2 years of age or the end of observation period, i.e. 31<sup>st</sup> December 2007, whichever came first. The main outcomes of interest were child immunisation status and timeliness of immunisation. Immunisation status was assessed in children aged 12-23 months while timeliness was assessed in all children aged less than 2 years. For assessing risk factors for child immunisation status and timeliness of immunisation, multivariable analyses were carried out using logistic regression models.

**Methods for child education**

The study population consisted of children between the ages of 6 and 14 years. The cut-off of six years was chosen because, as policy in Kenya, children are expected to join Standard 1 at the age of 6. Since primary education in Kenya has 8 grades, the earliest a child is expected to complete primary school is by age 14. Four outcomes were assessed in this chapter namely; school enrolment by age 7, grade repetition, interruption of schooling and grade-for-age.

The start of observation was marked by enumeration, attainment of age 6 or in-migration. The end of observation was marked by the child dropping out of school, death, outmigration, and attainment of 15 years of age or the end of observation period- 31<sup>st</sup> December 2007, whichever came first. For all four outcomes, logistic regression models were fitted to assess factors associated with the outcomes controlling for covariates.

All analyses were carried out in STATA 11 statistical software and statistical tests were two-tailed and 5% level of significance.

## **Results**

## 4. Levels, trends and risk factors for adult mortality

### 4.1 Introduction

Adult mortality in sub-Saharan Africa is very high and has been rising rapidly in the last two decades <sup>[8, 48, 58, 60, 230]</sup>. This has contributed to the decline in life expectancy in the region <sup>[59]</sup>. Prior to the recent changes in adult mortality, there was limited demographic research on adult mortality partly due to scarce data. A lot of investment in interventions and research on child health over the years has taken place, but only a few and committed interventions for adult health such as maternal health, HIV/AIDS, malaria and tuberculosis have been well funded, including related research. Gains made particularly in improving child health risk being eroded by prevalent poor adult health and premature death of the primary care givers of children. The coming on the scene of HIV/AIDS and resurgence of tuberculosis has contributed to the rise in adult mortality burden <sup>[4, 58, 90]</sup>. Also, the epidemiologic transition taking place in the developing world means that the non-communicable diseases are increasingly contributing to the overall burden of ill adult health, yet limited data are available to monitor these <sup>[68]</sup>.

Vital registration systems are poorly developed in both Kenya and many other African countries. The challenge of the recent changes in adult mortality have brought to the fore the need to have appropriate data to enable us to understand its dynamics and impact on households and communities. This has made the development and use of alternative sources of mortality data inevitable <sup>[8, 231]</sup>. A number of demographic methodologies to measure adult mortality using data other than that from the national censuses or vital registration have been developed <sup>[45]</sup>. However, there is no single agreed upon alternative data source and approach to measuring adult mortality in countries lacking proper vital registration or well implemented censuses <sup>[8, 48]</sup>. Furthermore, estimates given by the UN do not provide sub-national figures and yet countries tend to show great variability in the distribution of major causes of adult death.

Small area demographic surveillance systems are one alternative source of data that can be used to measure adult mortality and also assess its impact <sup>[231]</sup>. With these data, it is

possible to examine determinants of adult mortality but also individual and household level effects which other data sources may not permit. Surveillance system data allows for detailed examination of sub-populations such as slum dwellers whose health and social indicators are often not disaggregated and reported on in surveys or censuses. Slum sub-populations are assigned averages for “urban” population which often are derived from the more affluent population living in planned neighbourhoods. This is because slums tend to be under sampled due to the informal nature of housing structures and yet they constitute a large proportion of urban populations. This under representation not only has a potential of misrepresenting the health and social situation, but can also to mislead planning and allocation of resources.

This chapter explores adult mortality using data from the continuous health and demographic surveillance in two informal settlements. These data are used to estimate levels, trends and risk factors associated with adult mortality. The chapter lays the foundation for the rest of the thesis as adult mortality is the key explanatory variable for the various child health and social outcomes examined in later chapters.

## 4.2 Methods and data sources

### Age Standardisation

Differences in the age structure of different populations affect the observed crude death rates making comparisons across population invalid. I standardised the mortality rates to enable comparison between the two slums and other published mortality rates using two published age standards; the WHO global standard and the INDEPTH standard as shown in Table 1 <sup>[55, 232]</sup>.

Table 1: WHO global age distribution and INDEPTH age distribution for sub-Saharan Africa

Age group (years)	WHO global standard <sup>s</sup>	INDEPTH standard population*
0-4	0.0886	0.149418
4-9	0.0869	0.142497
10-14	0.0860	0.131040
15-19	0.0847	0.104564
20-24	0.0822	0.078289
25-29	0.0793	0.063646
30-34	0.0761	0.057554
35-39	0.0715	0.054802
40-44	0.0659	0.043456
45-49	0.0604	0.036307



50-54	0.0537	0.033110
55-59	0.0455	0.030741
60-64	0.0372	0.025024
65-69	0.0296	0.019660
70-74	0.0221	0.013432
75-79	0.0152	0.008473
80-84	0.0091	0.004740
85+	0.0064	0.001246

<sup>s</sup>*Standard global age distribution developed by WHO* <sup>[232]</sup>

<sup>\*</sup>*Standard age distribution developed by INDEPTH* <sup>[55]</sup>

Currently, the most widely-used age standard for standardising death rates is the WHO Global standard based on an average of observed population structures across the world, including sub-Saharan Africa, although it is not well represented due to lack of data <sup>[232]</sup>. It is widely believed that the WHO global standard and other earlier standards do not reflect the age structure in sub-Saharan Africa and tend to overestimate mortality for sub-Saharan Africa because they give more weight to age groups which naturally face higher risks of deaths particularly the elderly. Recently, the INDEPTH network, an umbrella organisation for the various demographic surveillance system sites, published life tables and generated a new age standard using data from the member sites <sup>[55]</sup>. I standardised mortality rates using both the WHO Global standard and the INDEPTH standard for comparison purposes with the population proportions of individuals aged 15 years and above readjusted to add up to 1 for each standard.

### Life table estimation

In order to be able to estimate the life expectancy, I used an abridged life table analysis for each gender starting from 15 years and having 85 years and above as the upper limit. The age-specific mortality rates were computed by dividing number of deaths in a given interval by the total number of person-years accumulated in the same interval. A hypothetical cohort of 100,000 was used as the radix and I also assumed that individuals who could have died in the interval, did so about half-way the interval ( ${}_na_x=n/2$ ). To be able to calculate the probability of dying in a given interval having been alive at the start of the interval, I used the following formula <sup>[233]</sup>:

$${}_nq_x = n * {}_nm_x / I + (n - {}_na_x) * {}_nm_x,$$

where  ${}_nq_x$  is the probability of dying in the interval;  $n$  is the length of the interval;  ${}_nm_x$  is the age-specific mortality rate; and  ${}_na_x$  is the average number of years lived by individuals who die in the interval. The probability of surviving in the interval was taken as the complement of  ${}_nq_x$ . From the radix, the number of individuals in the

subsequent intervals was computed as the number surviving in previous category multiplied by the corresponding probability of surviving the same interval. The number of person-years ( ${}_nL_x$ ) was calculated by multiplying number surviving interval ( $l_x$ ) by the interval length plus number who died in the interval ( ${}_nd_x$ ) multiplied by the average survival period of those who died in the interval ( ${}_na_x$ ). The number of person-years lived in the last age category ( ${}_nL_{85}$ ) was computed as the ratio of individuals living in that age group  $l_{85}$  to the age-specific mortality rate of the age category  ${}_m m_{85}$ .

### **Risk factors associated with survival in the slums**

Data were prepared for survival analysis with failure defined as death from any cause during the study period. Individuals who failed (died) were given a value of 1 and those who were censored were given a value of 0. I used Kaplan-Meier methods for descriptive analyses and for exploration of factors associated with risk of death I used Cox proportional hazard model and Piecewise exponential regression models. However because the parameter estimates from the Cox proportional hazard models were very similar to those obtained from the Piecewise exponential model, the rest of the analyses were carried out using Cox proportional hazard models. Interaction effects were also explored.

## **4.3 Results**

Table 2 gives a breakdown of number of deaths, person years, and the crude death rate by socio-demographic characteristics separately for females and males.

### **Crude death rates:**

Overall, the crude death rate for females was about 8 per 1000 person-years, while for males it was about 7.5 per 1000 person-years. For both sexes (Table 2), the crude death rate for Korogocho slum was about twice that for Viwandani slum. Among females, the CDR in Korogocho was over 10 per 1000 person years compared to about 6 per 1000 person-years in Viwandani. For males, the CDR among residents of Korogocho was about 11 per 1000 person-years compared to only 4.6 per 1000 person years in Viwandani. Crude death rates were highest among females of the Luo and Cushitic ethnic backgrounds while for males, mortality was highest among the Luo and Kikuyu ethnic groups. For both sexes, individuals with no formal education had crude death

rates of more than double that among individuals with primary or higher education. Individuals whose wealth status could not be ascertained had higher crude death rates on average for both sexes compared with the other wealth categories.

Table 2: Crude death rates per 1,000 person-years by socio-demographic characteristics

<b>Women</b>	Characteristics	Person-years	Deaths	CDR	(95% CI)
	Slum				
	Korogocho	38196.1	408	10.68	(9.69;11.77)
	Viwandani	39968.0	246	6.15	(5.43;6.97)
	Ethnicity				
	Kikuyu	27513.7	253	9.20	(8.13;10.40)
	Luhya	10394.0	78	7.50	(6.01;9.37)
	Luo	11709.9	163	13.92	(11.94;16.23)
	Kamba	18075.3	97	5.37	(4.40;6.55)
	Cushitic	4826.8	36	7.46	(5.38;10.34)
	Other	5644.4	27	4.78	(3.28;6.98)
	Wealth tertile				
	Poorest	23978.8	210	8.76	(7.65;10.03)
	Second	24680.5	147	5.96	(5.07;7.00)
	Wealthiest	28793.6	286	9.93	(8.85;11.15)
	Unknown	711.3	11	15.47	(8.56;27.93)
	Education level				
	None	5709.1	113	19.79	(16.46;23.80)
	Primary	50127.7	411	8.20	(7.44;9.03)
	Secondary	20410.1	113	5.54	(4.60;6.66)
	Unknown	1917.2	17	8.87	(5.51;14.26)
	<b>CDR for women</b>	<b>78164.1</b>	<b>654</b>	<b>8.37</b>	<b>(7.75;9.03)</b>
<b>Men</b>	Characteristics	Person-years	Deaths	CDR	(95% CI)
	Slum				
	Korogocho	46211.9	524	11.34	(10.41;12.35)
	Viwandani	68095.3	335	4.92	(4.42;5.48)
	Ethnicity				
	Kikuyu	33530.8	322	9.60	(8.61;10.71)
	Luhya	15270.6	99	6.48	(5.32;7.89)
	Luo	15952.7	236	14.79	(13.02;16.81)
	Kamba	33312.7	128	3.84	(3.23;4.57)
	Cushites	5207.5	34	6.53	(4.67;9.14)
	Other	11032.8	40	3.63	(2.66;4.94)
	Wealth tertile				
	Poorest	34521.2	289	8.37	(7.46;9.39)
	Second	41399.9	200	4.83	(4.21;5.55)
	Wealthiest	36913.1	350	9.48	(8.54;10.53)
	Unknown	1472.9	20	13.58	(8.76;21.05)
	Education level				
	None	3710.5	67	18.06	(14.21;22.94)
	Primary	64939.9	525	8.08	(7.42;8.81)
	Secondary	43054.6	241	5.60	(4.93;6.35)
	Unknown	2602.1	26	9.99	(6.80;14.68)
	<b>CDR for males</b>	<b>114307.1</b>	<b>859</b>	<b>7.51</b>	<b>(7.03;8.03)</b>

*CDR: Crude death rate; CI confidence interval*

### Mortality trends over time (2003-2007)

Although the study period was short (5 years), I tried to demonstrate a mortality trend given recent changes in mortality due to HIV/AIDS, which is a major cause of death. Mortality among women declined over the years but the trends are less clear among men. By slum of residence, mortality seems to have reduced among residents of Korogocho while there seem to be no clear change or trend among residents of Viwandani (Table 3). There also seem to be a clear downward trend in mortality rates among the Luo ethnic group. By wealth, it appears that the mortality among the wealthiest slightly decreased over time. Also, individuals whose wealth status was not known seem to have a marked downward trend in mortality over the years.

Table 3: Crude death rate trends for adults in Korogocho and Viwandani by socio-demographic characteristics

Variables	Mortality rates by calendar year per 1000 person-years				
	2003	2004	2005	2006	2007
Sex					
Female	11.15(9.59;12.97)	8.67(7.29;10.30)	8.16(6.86;9.71)	7.49(6.27;8.96)	6.60(5.47;7.97)
Male	7.93(6.86;9.17)	8.38(7.25;9.68)	6.76(5.77;7.91)	6.74(5.76;7.88)	7.81(6.76;9.03)
Slum					
Korogocho	13.96(12.24;15.92)	11.73(10.20;13.48)	10.52(9.09;12.17)	9.47(8.13;11.04)	9.77(8.39;11.37)
Viwandani	5.79(4.87;6.88)	5.76(4.80;6.91)	4.73(3.89;5.75)	5.12(4.25;6.16)	5.49(4.61;6.54)
Ethnicity					
Kikuyu	10.10(8.44;12.08)	9.26(7.68;11.17)	9.55(7.97;11.45)	8.04(6.61;9.78)	10.16(8.54;12.08)
Luhya	8.69(6.44;11.71)	8.90(6.60;12.00)	6.27(4.43;8.86)	5.47(3.80;7.87)	5.48(3.83;7.84)
Luo	22.74(19.08;27.09)	14.47(11.62;18.01)	11.63(9.12;14.83)	13.21(10.52;16.59)	10.11(7.76;13.16)
Kamba	3.74(2.74;5.09)	5.44(4.14;7.14)	3.79(2.76;5.21)	4.43(3.32;5.91)	4.58(3.46;6.06)
Cushites	4.37(2.19;8.74)	6.03(3.43;10.62)	9.64(6.22;14.94)	6.21(3.61;10.70)	8.30(5.16;13.36)
Other	4.44(2.63;7.49)	5.22(3.20;8.53)	2.72(1.41;5.23)	4.31(2.60;7.15)	3.54(2.06;6.10)
Wealth tertile					
Poorest	8.64(7.14;10.45)	9.62(7.94;11.66)	8.44(6.91;10.31)	7.69(6.27;9.45)	8.35(6.88;10.14)
Middle	6.13(4.92;7.63)	5.08(3.97;6.49)	3.81(2.89;5.02)	5.49(4.37;6.90)	5.72(4.59;7.13)
Wealthiest	12.63 (10.78;14.80)	10.20(8.60; 12.09)	9.77(8.23;11.58)	7.99(6.62;9.64)	8.14(6.75;9.82)
Unknown	19.08(10.56;34.45)	30.76(17.47;54.17)	8.84(2.85;27.41)	8.35(2.69;25.90)	3.85(0.96;15.41)
Education					
No education	21.92(16.47;29.18)	16.55(11.88;23.05)	16.26(11.44;23.13)	18.04(12.69;25.65)	23.44(16.90;32.49)
Primary	9.83(8.60;11.24)	8.79(7.64;10.13)	7.83(6.77;9.06)	7.01(6.02;8.15)	7.40(6.40;8.57)
Secondary/higher	6.27(5.05;7.79)	5.98(4.74;7.55)	5.06(3.95;6.47)	5.37(4.24;6.80)	5.22(4.12;6.61)
Unknown	6.44(2.89;14.34)	15.25(9.03;25.74)	6.73(3.02;14.98)	11.07(5.96;20.57)	8.00(3.81;16.78)
Total	9.21(8.29;10.22)	8.49(7.60;9.49)	7.33(6.52;8.23)	7.05(6.27;7.93)	7.31(6.52;8.20)
Score test for trend of rates	RR 0.94	95% CI (0.90-0.97)	Chi2 13.30	P- value 0.0003	

By education, mortality reduced in all categories between 2003 and 2005 but stalled and appears to have increased between 2005 and 2007 among those with no formal education and those whose education status was not known.

#### Age-specific mortality rates:

Table 4 shows that, as expected, the adult mortality rate increased with age. Overall, mortality among women was about 28% higher than that recorded among men, however there were a few exceptions of age groups where mortality among women was lower than that among males including the age categories 15-19 years, 65-69, 75-79 and 85 years and above. Significant differences in age-specific mortality rates by gender were only seen in the age group 15-19 in which mortality was higher among males than females; and 25-29, 30-34 and 35-39 in which mortality was higher among females.

Table 4: Age- and Sex-specific mortality rate per 1000 person-years for Korogocho and Viwandani slums combined

Age group	Women				Men				RR	(95% CI)
	Person-years	Deaths	ASMR	(95% CI)	Person-years	Deaths	ASMR	(95% CI)		
15-19yrs	12263.5	21	1.71	(1.12;2.63)	10472.8	42	4.01	(2.96;5.43)	0.43	(0.25;0.72)
20-24yrs	20971.3	77	3.67	(2.94;4.59)	21216.1	76	3.58	(2.86;4.49)	1.02	(0.75;1.41)
25-29yrs	16968.9	118	6.95	(5.81;8.33)	25551.1	112	4.38	(3.64;5.28)	1.59	(1.22;2.05)
30-34yrs	10349.9	140	13.53	(11.46;15.96)	19378.3	140	7.22	(6.12;8.53)	1.87	(1.48;2.37)
35-39yrs	6540.1	87	13.3	(10.78;16.41)	13296.4	111	8.35	(6.93;10.06)	1.59	(1.2;2.11)
40-44yrs	4367.0	59	13.51	(10.47;17.44)	9499.3	121	12.74	(10.66;15.22)	1.06	(0.78;1.45)
45-49yrs	2331.7	34	14.58	(10.42;20.41)	6331.4	71	11.21	(8.89;14.15)	1.3	(0.86;1.96)
50-54yrs	1593.7	32	20.08	(14.2;28.39)	3860.4	60	15.54	(12.07;20.02)	1.29	(0.84;1.98)
55-59yrs	947.8	18	18.99	(11.97;30.14)	2285.0	42	18.38	(13.58;24.87)	1.03	(0.59;1.79)
60-64yrs	637.9	15	23.52	(14.18;39.01)	1100.0	19	17.27	(11.02;27.08)	1.36	(0.69;2.68)
65-69yrs	356.0	8	22.47	(11.24;44.93)	570.1	22	38.59	(25.41;58.61)	0.58	(0.26;1.31)
70-74yrs	307.1	15	48.84	(29.45;81.02)	330.3	15	45.41	(27.38;75.32)	1.08	(0.53;2.2)
75-79yrs	221.7	5	22.55	(9.39;54.19)	188.4	9	47.78	(24.86;91.82)	0.47	(0.16;1.41)
80-84yrs	155.5	10	64.32	(34.61;119.54)	138.7	7	50.47	(24.06;105.87)	1.27	(0.49;3.35)
85+yrs	152.0	15	98.66	(59.48;163.65)	88.8	12	135.16	(76.76;238)	0.73	(0.34;1.56)
Total	78164.1	654	8.37	(7.75;9.03)	114307.1	859	7.51	(7.03;8.03)	1.11	(1.01;1.23)

RR=Rate ratio: females to males=1.28 (1.15;1.42) (controlling for age):  $X^2=22.49$ ,  $P$  value <0.0001

ASMR: Age-specific mortality rate; CI confidence interval

## Age-adjusted mortality rates:

Table 5 shows age adjusted death rates alongside the crude death rates. For comparison purposes, I provided the adjusted rates using both the INDEPTH and WHO global standard.

**Table 5: Age adjusted mortality rates per 1,000 (using WHO world standard & INDEPTH standard).**

Variables	Person-years	CDR	WHO standard		INDEPTH standard	
			Adjusted death rate	(95% CI)	Adjusted death rate	(95% CI)
<b>Slum</b>						
Korogocho	38198	10.68	15.56	(13.69;17.43)	13.26	(11.74;14.78)
Viwandani	39970	6.16	16.73	(11.71;21.76)	13.69	(9.71;17.66)
<b>Ethnicity</b>						
Kikuyu	27514	9.20	14.47	(12.30;16.65)	12.07	(10.31;13.84)
Luhya	10394	7.50	15.68	(8.39;22.97)	13.78	(7.55;20.01)
Luo	11711	13.92	28.55	(11.29;45.82)	24.97	(11.29;38.66)
Kamba	18077	5.37	12.78	(7.75;17.82)	10.56	(6.75;14.37)
Cushites	4799	6.67	9.25	(5.66;12.85)	8.09	(5.11;11.07)
Other	5673	5.46	11.02	(3.52;18.52)	8.72	(2.83;14.61)
<b>Wealth tertile</b>						
First	23979	8.76	14.84	(12.16;17.51)	12.66	(10.46;14.86)
Second	24681	5.96	14.44	(8.80;20.07)	11.80	(7.28;16.31)
Third	28795	9.93	15.83	(13.30;18.35)	13.18	(11.22;15.14)
Unknown	711	15.47	38.19	(0.00; --- )	33.46	(0.00; --- )
<b>Education level</b>						
None	5707	19.80	16.97	(13.78;20.16)	14.35	(11.46;17.24)
Primary	50128	8.20	15.03	(11.93;18.13)	12.99	(10.56;15.42)
Secondary	20410	5.54	6.15	(0.00; --- )	5.54	(0.00; --- )
Unknown	1917	8.87	12.82	(6.50;19.15)	11.21	(5.78;16.65)
<b>Total for females</b>	<b>78168</b>	<b>8.37</b>	<b>14.71</b>	<b>(13.05;16.36)</b>	<b>12.32</b>	<b>(11.00;13.65)</b>
<b>Males</b>						
<b>Slum</b>						
Korogocho	46212	11.34	17.19	(15.21;19.18)	14.56	(13.00;16.12)
Viwandani	68097	4.92	9.91	(6.59;13.23)	7.71	(5.96;9.46)
<b>Ethnicity</b>						
Kikuyu	33530	9.60	15.65	(13.26;18.03)	13.25	(11.36;15.13)
Luhya	15272	6.48	10.95	(6.59;15.31)	9.87	(6.28;13.47)
Luo	15953	14.79	24.50	(14.01;34.99)	20.56	(13.05;28.07)
Kamba	33313	3.84	9.52	(5.98;13.06)	7.57	(5.27;9.88)
Cushites	5207	6.53	10.65	(6.70;14.59)	8.06	(5.06;11.05)
Other	11033	3.63	7.59	(3.37;11.81)	6.35	(3.06;9.64)
<b>Wealth tertile</b>						
Poorest	34522	8.37	15.19	(12.51;17.87)	12.62	(10.58;14.66)
Second	41399	4.83	7.78	(5.59;9.97)	6.85	(5.10;8.61)
Wealthiest	36913	9.48	15.55	(13.14;17.97)	12.92	(11.07;14.76)
Unknown	1473	13.58	16.28	(8.35;24.21)	14.13	(7.52;20.74)
<b>Education level</b>						
None	3710	18.06	16.22	(12.20;20.24)	14.09	(10.19;17.99)
Primary	64941	8.08	14.28	(11.92;16.63)	11.83	(10.17;13.49)
Secondary	43056	5.60	14.49	(8.96;20.01)	12.58	(8.10;17.06)
Unknown	2601	10.00	15.26	(8.21;22.31)	11.41	(6.60;16.22)
<b>Total for males</b>	<b>114309</b>	<b>7.52</b>	<b>13.74</b>	<b>(12.22;15.27)</b>	<b>11.31</b>	<b>(10.18;12.45)</b>
<b>Total for females &amp; males</b>	<b>192477</b>	<b>7.86</b>	<b>13.83</b>	<b>(12.75;14.91)</b>	<b>11.48</b>	<b>(10.64;12.32)</b>



By sex, even after controlling for age, mortality among females remained higher than that among males. For both females and males, the adjusted mortality rates were higher than the corresponding crude death rates across all variables. Also, generally the adjusted rates given by the WHO global standard were higher than those given by the INDEPTH standard. Among females, the apparently big difference in death rates by slum was reduced to a very small difference after adjustment using either standard, with slightly higher mortality in Viwandani. The difference observed in the crude rate by slum among males however persists after adjustment using either standard. By ethnic group, the Luo, Luhya and Kikuyu women and men had higher adjusted death rates. Females and males with no formal education had the highest adjusted death rate by either standard. Overall, the WHO global standard resulted into an adjusted mortality rate of about 20.5% higher than that estimated using the INDEPTH standard.

## Life expectancy

Table 6 shows an abridged period life table for the two slums combined presented by sex. The life table notation in Table 6 is defined below:

*${}_nD_x$ -Number of deaths in the interval;  ${}_nm_x$ - Age specific mortality rate in the interval;  ${}_nq_x$ - conditional probably of dying in the interval;  ${}_np_x$ - probability of surviving in the interval;  $l_x$ - number surviving at the beginning of the interval;  ${}_nd_x$ - number of those who died in the interval;  ${}_nL_x$ - Number of person years;  $T_x$ - number of person years lived at age  $x$  and older and  $e_x$ - life expectancy at age  $x$ .*

Life expectancy at the age of 15 years for males was 44 years, about two years more than that for females (41 years).

Table 6: Life table from Nairobi Urban Health and Demographic Surveillance site 2003-2007

Age group	Person-years	$nD_x$	$nm_x$	$nq_x$	$np_x$	$lx$	$nd_x$	$nL_x$	$T_x$	$e_x$
<b>Females</b>										
15-19yrs	12263.5	21	0.001712	0.012843	0.987157	100,000	1,284	496,789	4,140,680	41.4
20-24yrs	20971.3	77	0.003672	0.027538	0.972462	98,716	2,718	486,783	3,643,891	36.9
25-29yrs	16968.9	118	0.006954	0.052154	0.947846	95,997	5,007	467,470	3,157,109	32.9
30-34yrs	10349.9	140	0.013527	0.101451	0.898549	90,991	9,231	431,876	2,689,639	29.6
35-39yrs	6540.1	87	0.013303	0.099770	0.900230	81,760	8,157	388,405	2,257,763	27.6
40-44yrs	4367.0	59	0.013510	0.101328	0.898672	73,602	7,458	349,367	1,869,358	25.4
45-49yrs	2331.7	34	0.014582	0.109364	0.890636	66,144	7,234	312,638	1,519,991	23.0
50-54yrs	1593.7	32	0.020079	0.150592	0.849408	58,911	8,871	272,375	1,207,353	20.5
55-59yrs	947.8	18	0.018991	0.142432	0.857568	50,039	7,127	232,378	934,978	18.7
60-64yrs	637.9	15	0.023516	0.176367	0.823633	42,912	7,568	195,639	702,600	16.4
65-69yrs	356.0	8	0.022471	0.168531	0.831469	35,344	5,957	161,827	506,961	14.3
70-74yrs	307.1	15	0.048842	0.366316	0.633684	29,387	10,765	120,024	345,134	11.7
75-79yrs	221.7	5	0.022553	0.169151	0.830849	18,622	3,150	85,236	225,110	12.1
80-84yrs	155.5	10	0.064320	0.482401	0.517599	15,472	7,464	58,702	139,874	9.0
85+yrs	152.0	15	0.098660	1.000000	0.000000	8,008	8,008	81,172	81,172	10.1
<b>Males</b>										
15-19yrs	10472.8	42	0.004010	0.030078	0.969922	100,000	3,008	492,481	4,385,651	43.9
20-24yrs	21216.1	76	0.003582	0.026866	0.973134	96,992	2,606	478,446	3,893,171	40.1
25-29yrs	25551.1	112	0.004383	0.032875	0.967125	94,386	3,103	464,174	3,414,724	36.2
30-34yrs	19378.3	140	0.007225	0.054184	0.945816	91,283	4,946	444,052	2,950,550	32.3
35-39yrs	13296.4	111	0.008348	0.062611	0.937389	86,337	5,406	418,172	2,506,498	29.0
40-44yrs	9499.3	121	0.012738	0.095533	0.904467	80,932	7,732	385,329	2,088,326	25.8
45-49yrs	6331.4	71	0.011214	0.084105	0.915895	73,200	6,156	350,609	1,702,997	23.3
50-54yrs	3860.4	60	0.015542	0.116567	0.883433	67,043	7,815	315,680	1,352,388	20.2
55-59yrs	2285.0	42	0.018381	0.137854	0.862146	59,228	8,165	275,730	1,036,708	17.5
60-64yrs	1100.0	19	0.017272	0.129541	0.870459	51,064	6,615	238,781	760,979	14.9
65-69yrs	570.1	22	0.038593	0.289447	0.710553	44,449	12,866	190,080	522,198	11.7
70-74yrs	330.3	15	0.045410	0.340578	0.659422	31,583	10,757	131,025	332,118	10.5
75-79yrs	188.4	9	0.047776	0.358319	0.641681	20,827	7,463	85,477	201,094	9.7
80-84yrs	138.7	7	0.050474	0.378555	0.621445	13,364	5,059	54,173	115,617	8.7
85+yrs	88.8	12	0.135164	1.000000	0.000000	8,305	8,305	61,444	61,444	7.4

After age 49, life expectancy for females becomes higher than that for males and by age 60, life expectancy among females is 16.4 years compared to 14.9 years among males. While females who have attained age 84 can expect to live on average more than 10 years, males on average would expect to live for about 7.4 years.

Life expectancy estimates from this study and 10 other DSS sites published earlier on are compared. Detailed table in Appendix II <sup>[55]</sup>. Where there was more than one site from a given country, the site with longest follow up period was chosen. Generally, life expectancy at 15 at the Nairobi DSS was lower than majority of the other sites with exception of Bandim and Manhica (females) sites. With the exception of Agincourt site, life expectancy at 15 was less than 60 years in all sites. NUHDSS site is also the only site where life expectancy at 15 among males is higher than that among females. At the age of 60, females at NUHDSS expect to live an extra 16.4 years while those of Agincourt expect to live about 24 years and those of Bandim site expect to live for only 10.4 years. At the age of 60 years, males at NUHDSS expect to live for 14.9 years compared to 20.2 at Agincourt and only 7.7 years at Bandim. Life expectancy at 84 years is generally higher at the Nairobi DSS compared with majority of the other sites with the exception of Agincourt site, but due to the small numbers in this category, these estimates are not very stable.

### **Conditional probability of dying between 15 and 60 years of age**

Results in Table 7 show that the probability of a 15 year old female dying before reaching her 60<sup>th</sup> birthday was 0.571 (571 per 1000) and that for males was 0.489 (489 per 1000). Before the age of 25 years, the cumulative probability of dying was higher among males however this changes at the age of 25 years whereby the cumulative probability of death among females becomes higher than that for males and remained that way till old age.

Table 7: Adult mortality: Probability of dying before reaching 60 years after one has lived up to 15 years

Age group	Person-years	Deaths	${}_n m_x$	${}_n q_x$	${}_n p_x$	Cumulative survival probability	Cumulative probability of death
<b>Females</b>							
15-19yrs	12263.5	21	0.001712	0.012843	0.987157	0.987157	0.012843
20-24yrs	20971.3	77	0.003672	0.027538	0.972462	0.959973	0.040027
25-29yrs	16968.9	118	0.006954	0.052154	0.947846	0.909906	0.090094
30-34yrs	10349.9	140	0.013527	0.101451	0.898549	0.817595	0.182405
35-39yrs	6540.1	87	0.013303	0.099770	0.900230	0.736024	0.263976
40-44yrs	4367.0	59	0.013510	0.101328	0.898672	0.661444	0.338556
45-49yrs	2331.7	34	0.014582	0.109364	0.890636	0.589106	0.410894
50-54yrs	1593.7	32	0.020079	0.150592	0.849408	0.500391	0.499609
55-59yrs	947.8	18	0.018991	0.142432	0.857568	0.429120	<b>0.570880</b>
60-64yrs	637.9	15	0.023516	0.176367	0.823633	0.353437	0.646563
65-69yrs	356.0	8	0.022471	0.168531	0.831469	0.293872	0.706128
70-74yrs	307.1	15	0.048842	0.366316	0.633684	0.186222	0.813778
75-79yrs	221.7	5	0.022553	0.169151	0.830849	0.154722	0.845278
80-84yrs	155.5	10	0.064320	0.482401	0.517599	0.080084	0.919916
85+yrs	152.0	15	0.098660	1.000000	0.000000	0.000000	1.000000
<b>Males</b>							
Age group	Person-years	Deaths	${}_n m_x$	${}_n q_x$	${}_n p_x$	Cumulative survival probability	Cumulative probability of death
15-19yrs	10472.8	42	0.004010	0.030078	0.969922	0.969922	0.030078
20-24yrs	21216.1	76	0.003582	0.026866	0.973134	0.943864	0.056136
25-29yrs	25551.1	112	0.004383	0.032875	0.967125	0.912835	0.087165
30-34yrs	19378.3	140	0.007225	0.054184	0.945816	0.863374	0.136626
35-39yrs	13296.4	111	0.008348	0.062611	0.937389	0.809317	0.190683
40-44yrs	9499.3	121	0.012738	0.095533	0.904467	0.732000	0.268000
45-49yrs	6331.4	71	0.011214	0.084105	0.915895	0.670435	0.329565
50-54yrs	3860.4	60	0.015542	0.116567	0.883433	0.592285	0.407715
55-59yrs	2285.0	42	0.018381	0.137854	0.862146	0.510636	<b>0.489364</b>
60-64yrs	1100.0	19	0.017272	0.129541	0.870459	0.444488	0.555512
65-69yrs	570.1	22	0.038593	0.289447	0.710553	0.315832	0.684168
70-74yrs	330.3	15	0.045410	0.340578	0.659422	0.208267	0.791733
75-79yrs	188.4	9	0.047776	0.358319	0.641681	0.133641	0.866359
80-84yrs	138.7	7	0.050474	0.378555	0.621445	0.083050	0.916950
85+yrs	88.8	12	0.135164	1.000000	0.000000	0.000000	1.000000

Figure 5(a) shows a graphical representation of the conditional probability of dying between the age of 15 and 60 years. As noted earlier, the probability of dying before age of 25 years is higher among males. However, after the age of 25 years, the probability of dying among females exceeds that of males through to the age of 60 as seen in Figure 5(a).

Figure 5(a): Cumulative probability of dying among individuals aged between 15 and 60 years.

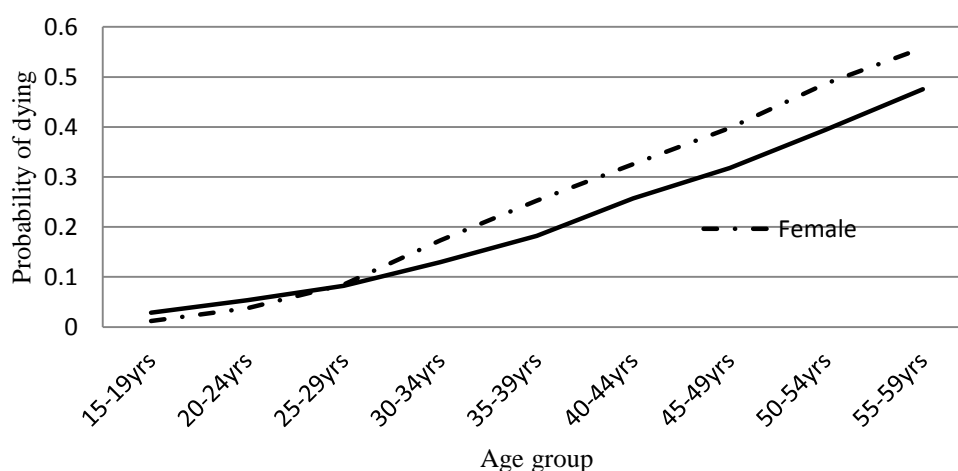
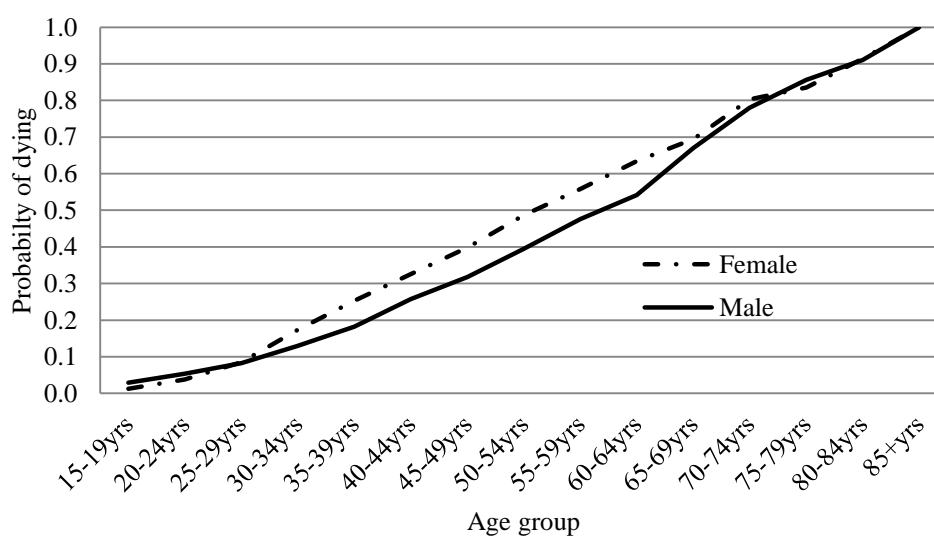


Figure 5 (b) shows the conditional probability of death for all adults. The probability of death for females and males converges at about 65 years of age and remain nearly the same through the age of 85 years. This deviation from the expected trend of higher probability of death among males compared with females may be related to excess mortality due to HIV/AIDS among women and maternal causes which are still prevalent in this setting.

Figure 5 (b): Cumulative probability of dying among individuals aged between 15 and over.

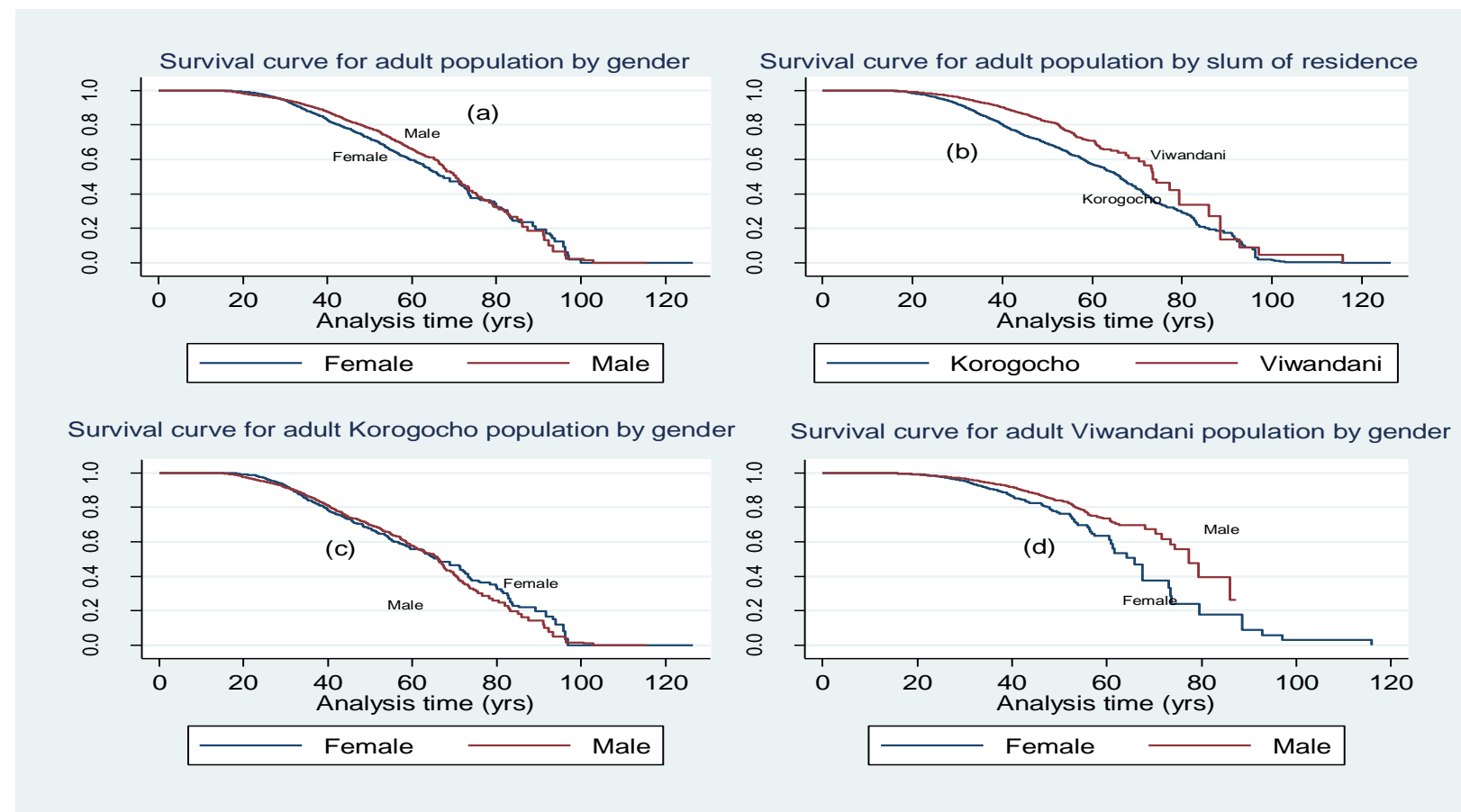


### Survival by some socio-demographic characteristics

From the foregoing results, it appears that there is a survival disadvantage for females compared with males. There also seem to be differences in survival by slum of residence with residents of Korogocho appearing to have higher mortality. Figure 6 shows Kaplan-Meier plots by gender and slum.

The log-rank test for equality of survivor functions by gender-graph (a) gave a chi-squared=22.8, p value <0.001; by slum-graph (b) chi-squared=128.7; p value <0.001; by gender for Korogocho slum- graph (c) chi squared=0.13; p value=0.719 and by gender for Viwandani slum-graph (d) chi squared=31.1; p value <0.001. These results show that there are significant differences in survival between males and females with males having a survival advantage while residents of Viwandani had better survival than those of Korogocho.

Figure 6: Survival curves by gender and by slum



On stratifying on slum, there was no significant difference in survival by gender in Korogocho slum while in Viwandani slum the difference in survival between males and females was even more exaggerated with males having much better survival.

### Factors associated with adult survival in the slums

Table 8 shows results from Cox proportional hazard models. The risk of death among males was lower than that among females. The hazard ratio increased from 0.78 to 0.87 after controlling for other covariates and was highly significant (p value=0.009). The risk of death among residents of Viwandani slum was about 46% lower than of Korogocho slum and this reduced to 26% lower after controlling for other covariates. Compared with the Kikuyu ethnic group, the risk of death among the Luhya, Kamba, and Cushites was lower. On the other hand the Luo ethnic group had about 77% higher risk of death compared with the Kikuyu after controlling for other covariates.

Table 8: Factors associated with risk of death in the slums

Variables	Unadjusted hazard ratios			Adjusted hazard ratios		
	Hazard Ratio	(95% CI)	P value	Hazard Ratio	(95% CI)	P value
Gender						
Females	1.00			1.00		
Males	0.78	(0.70;0.86)	<0.001	0.87	(0.78;0.96)	0.009
Slum						
Korogocho	1.00			1.00		
Viwandani	0.54	(0.49;0.60)	<0.001	0.74	(0.62;0.88)	0.001
Ethnicity						
Kikuyu	1.00			1.00		
Luhya	0.81	(0.68;0.96)	0.016	0.83	(0.70;0.99)	0.038
Luo	1.85	(1.62;2.11)	<0.001	1.77	(1.55;2.03)	<0.001
Kamba	0.52	(0.45;0.61)	<0.001	0.60	(0.51;0.70)	<0.001
Cushites	0.69	(0.53;0.88)	0.004	0.61	(0.47;0.80)	<0.001
Others	0.49	(0.38;0.63)	<0.001	0.56	(0.43;0.72)	<0.001
Wealth tertile						
Poorest	1.00			1.00		
Middle	0.65	(0.57;0.75)	<0.001	0.88	(0.74;1.04)	0.124
Wealthiest	1.12	(0.99;1.26)	0.061	0.82	(0.71;0.93)	0.003
Unknown	1.91	(1.33;2.75)	<0.001	1.98	(1.37;2.85)	<0.001
Education						
No education	1.00			1.00		
Primary	0.93	(0.77;1.12)	0.451	0.90	(0.74;1.09)	0.280
Secondary & higher	0.60	(0.49;0.74)	<0.001	0.66	(0.53;0.83)	<0.001
Unknown	0.85	(0.61;1.20)	0.354	0.94	(0.67;1.33)	0.733
Calendar year						
2003	1.00			1.00		
2004	0.91	(0.78;1.07)	0.252	0.91	(0.78;1.06)	0.213
2005	0.78	(0.67;0.92)	0.002	0.78	(0.67;0.92)	0.003
2006	0.73	(0.62;0.86)	<0.001	0.74	(0.63;0.87)	<0.001
2007	0.76	(0.65;0.89)	0.001	0.78	(0.67;0.91)	0.002



The wealthiest individuals had 18% lower risk of death compared with the poorest, while individuals with secondary or higher education had 34% lower risk of death compared with those with no formal education. Individuals whose wealth status was not ascertained had a markedly higher risk of dying (about 2 times) compared with the poorest. There seems to be a general trend of mortality decreasing over the years with significantly lower risk of death between 2005 and 2007. Given that there was some departure from the proportionality assumption of the Cox model, a piecewise exponential model was fitted for comparison, Appendix III. Because age was used as the “time” variable in the measuring of survival time, it could not be used as a covariate in the Cox proportional model as it is stratified on in the regression. Estimates for age from the Piecewise exponential model show very slight increase risk of death with age. The other estimates from the two models are very similar in magnitude and level of significance indicating that the proportionality assumption was after all not seriously violated.

### **Stratification on gender and slum of residence**

Because gender has a strong influence on risk of death, I stratified on gender and re-estimated the model. Results in Table 9 show that while risk of death significantly differs by slum among males, it does not differ much by slum among females. Among females, the risk of death significantly differs by ethnicity. The Luo were about 95% more likely to die while the Kamba were about 25% less likely to die compared with the Kikuyu. Among males, there were numerous differences by ethnicity. The Luhya and Kamba were about 28% and 49%, less likely to die than the Kikuyu respectively while the Luo were over 60% more likely to die as were Kikuyu. Both males and females with secondary or higher education were at least 35% less likely to die compared with those with no formal education. Risk of death among females of unknown wealth status was approximately twice as likely to die compared with the poorest. For males, the wealthiest were about 29% less likely to die compared with the poorest while those of unknown wealth status were nearly two times more likely to die compared with the poorest. While mortality consistently and significantly decreased since 2003 among females, there were no significant decreases among males.

Table 9: Hazard rates from Cox proportional hazard models by sex

Variables	Females			Males		
	Hazard Ratio	(95% CI)	P value	Hazard Ratio	(95% CI)	P value
Slum						
Korogocho	1.00			1.00		
Viwandani	0.94	(0.72;1.23)	0.634	0.62	(0.49;0.77)	<0.001
Ethnicity						
Kikuyu	1.00			1.00		
Luhya	0.99	(0.76;1.29)	0.940	0.72	(0.58;0.91)	0.006
Luo	1.95	(1.58;2.41)	<0.001	1.63	(1.37;1.95)	<0.001
Kamba	0.75	(0.59;0.97)	0.026	0.51	(0.41;0.63)	<0.001
Cushites	0.72	(0.49;1.05)	0.092	0.49	(0.33;0.73)	<0.001
Others	0.70	(0.47;1.05)	0.088	0.48	(0.34;0.67)	<0.001
Wealth tertile						
Poorest	1.00			1.00		
Middle	0.86	(0.67;1.12)	0.273	0.89	(0.71;1.11)	0.287
Wealthiest	0.96	(0.78;1.18)	0.702	0.71	(0.60;0.85)	<0.001
Unknown	2.05	(1.12;3.78)	0.021	1.93	(1.23;3.05)	0.005
Education						
No education	1.00			1.00		
Primary	0.88	(0.67;1.15)	0.352	0.83	(0.61;1.12)	0.225
Secondary & higher	0.63	(0.45;0.86)	0.005	0.65	(0.47;0.90)	0.009
Unknown	0.77	(0.45;1.29)	0.318	1.07	(0.66;1.72)	0.789
Calendar year						
2003	1.00			1.00		
2004	0.76	(0.60;0.96)	0.023	1.03	(0.83;1.26)	0.812
2005	0.75	(0.59;0.94)	0.014	0.81	(0.65;1.00)	0.056
2006	0.65	(0.51;0.83)	<0.001	0.81	(0.66;1.01)	0.062
2007	0.59	(0.46;0.75)	<0.001	0.94	(0.77;1.16)	0.569

Results seen earlier in Figure 6 and Table 8 showed significant differences in survival by slum. To explore further, I stratified on slum and re-estimated the models. Results in Table 10 shows that there were no significant differences in risk of death by gender in Korogocho slum, while in Viwandani slum the risk of death was lower among males by about 31%.

Table 10: Hazard rates from Cox proportional hazard models by slum

Variables	Korogocho			Viwandani		
	Hazard Ratio	(95% CI)	P value	Hazard Ratio	(95% CI)	P value
Gender						
Female	1.00			1.00		
Male	0.99	(0.87;1.13)	0.893	0.69	(0.58;0.82)	<0.001
Ethnicity						
Kikuyu	1.00			1.00		
Luhya	0.71	(0.57;0.89)	0.003	1.02	(0.78;1.33)	0.901
Luo	1.67	(1.42;1.95)	<0.001	1.88	(1.42;2.48)	<0.001
Kamba	0.62	(0.45;0.84)	0.002	0.64	(0.52;0.79)	<0.001
Cushites	0.55	(0.41; 0.75)	<0.001	0.83	(0.45;1.53)	0.545
Others	0.61	(0.40;0.93)	0.023	0.57	(0.41;0.80)	0.001
Wealth tertile						
Poorest	1.00			1.00		

Middle	---	---	---	0.86	(0.72;1.02)	0.086
Wealthiest	0.83	(0.72;0.96)	0.011	0.80	(0.52;1.23)	0.307
Unknown	2.52	(1.58;4.02)	<0.001	1.49	(0.83;2.68)	0.177
Education						
No education	1.00			1.00		
Primary	0.94	(0.75;1.19)	0.622	0.64	(0.43;0.96)	0.031
Secondary & higher	0.72	(0.55;0.95)	0.019	0.48	(0.32;0.73)	0.001
Unknown	0.93	(0.58;1.48)	0.748	0.78	(0.44;1.36)	0.380
Calendar year						
2003	1.00			1.00		
2004	0.85	(0.70;1.03)	0.102	1.00	(0.78;1.30)	0.970
2005	0.77	(0.63;0.93)	0.009	0.81	(0.62;1.06)	0.120
2006	0.67	(0.55;0.83)	<0.001	0.86	(0.66;1.11)	0.253
2007	0.69	(0.57;0.85)	<0.001	0.92	(0.72;1.18)	0.508

In Korogocho slum the Luhya were about 29% less likely to die compared with Kikuyu but this was not significant in Viwandani. The Luo in Korogocho were about 67% more likely to die while in Viwandani they were about 88% more likely to die as compared with Kikuyu. The Kamba in Korogocho were about 38% less likely to die while those in Viwandani were about 36% compared with the Kikuyu. The Cushites in Korogocho were 45% less likely to die compared with the Kikuyu but there were no significant differences in Viwandani in the same ethnic group. The wealthiest individuals in Korogocho were about 27% less likely to die compared with the poorest. There was no significant difference in risk of death by wealth status in Viwandani. Risk of death among individuals with secondary or higher education was about 28% lower in Korogocho and about 52% lower in Viwandani. In Korogocho there was a significant down ward trend in risk of death over the years since 2003 while there was no clear trend in Viwandani.

Figure 7(a) and (b) show trends in risk of death by gender and slum respectively controlling for other covariates. Generally there was a downward trend with a sharper decline among females and residents of Korogocho. However for males, it is difficult to assess whether some decline occurred and whether this trend was reversed toward the end of the period of observation or whether the fluctuations reflect issues of data quality. Figure 7 (c) shows trend in the risk of death stratified on both gender and slum of residence and controlling for other covariates. The figure shows that the decline in risk of death was greatest among females of Korogocho followed by females of Viwandani. The downward trend of risk of death among males started in 2004, however

this trend seems to have stalled and actually started reversing in 2005 in Viwandani and 2006 in Korogocho.

Figure 7(a): Mortality trends by sex after controlling for covariates

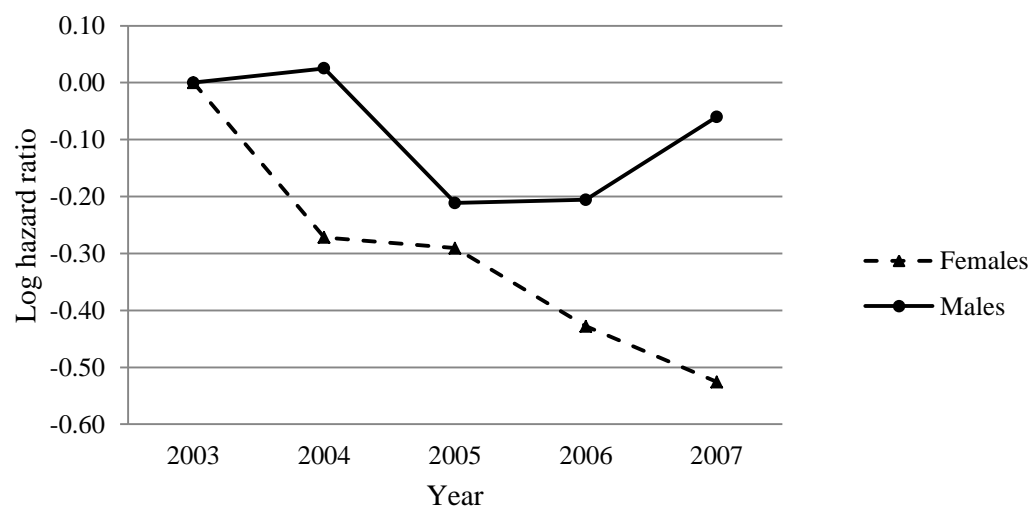


Figure 7 (b): Mortality trends by slum after controlling for covariates

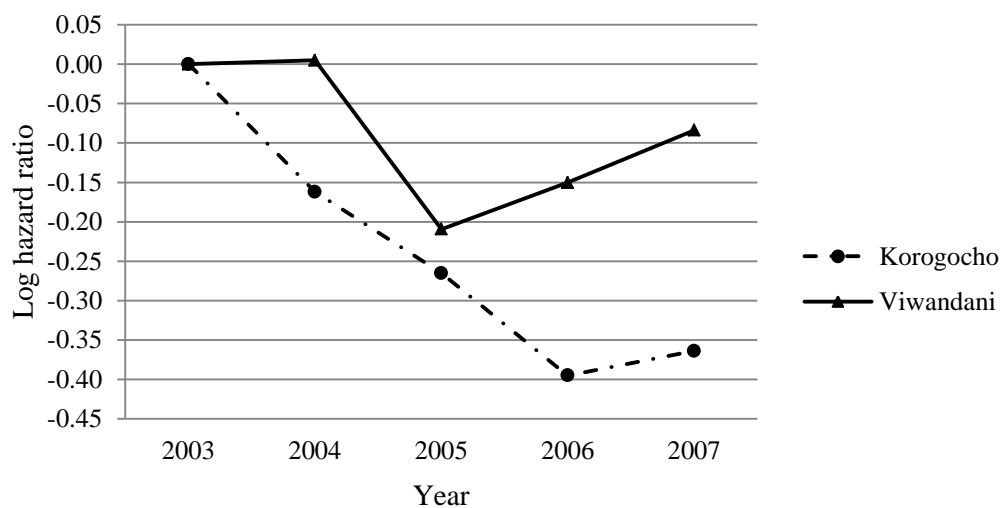
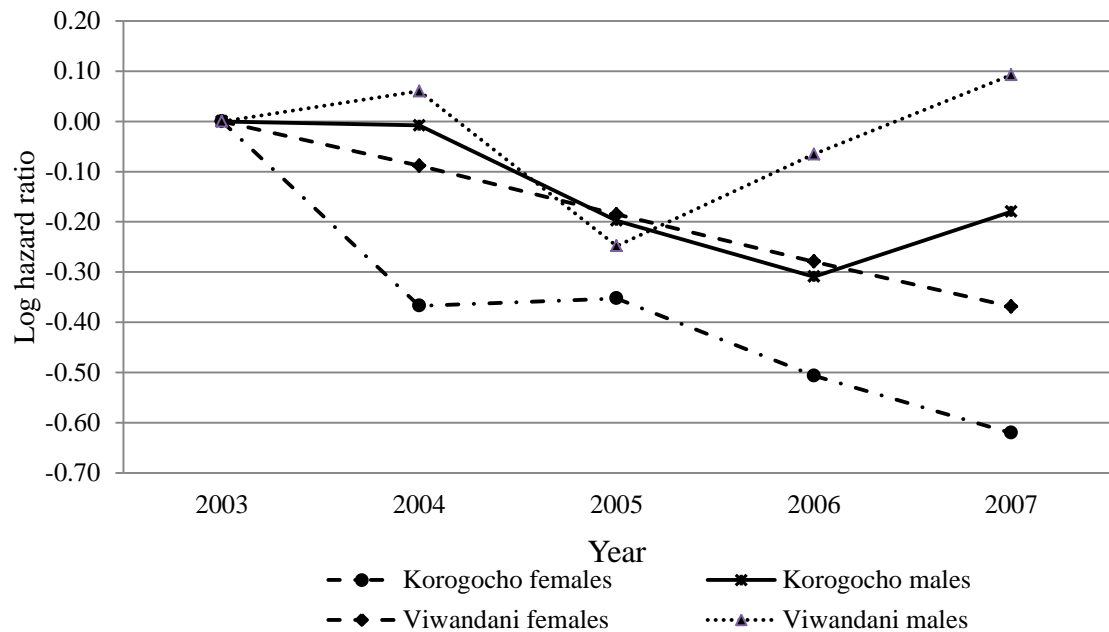


Figure 7 (c): Mortality trends stratified on gender and slum after controlling for other covariates



### Exploring interaction effects

Several interaction terms were added to the model one at a time. Those that were significant at 5% level of significance when added individually to the model were added to the final model. The interactions that were significant include: gender and slum; gender and calendar year and slum of residence and education. The likelihood-ratio test showed that the model with interactions was a better fit compared with that without ( $\chi^2=24.94$ ,  $p$  value $<0.0016$ ). Table 11 shows the model without (Model I) and those with interaction terms (Model II-VI), for ease of comparison.

Model II has the interaction term of gender and slum. The results show that the risk of death among males of Korogocho was not different from that of females of Korogocho while females of Viwandani had a 10% lower risk compared with females in Korogocho but not significant. Males of Viwandani had about 37% lower risk of dying compared with females of Korogocho.

Results in model III show that, mortality among males in 2003 was 30% lower than that among females in the same year. Among females, risk of death consistently reduced

over the years to up to 41% lower in 2007 compared with 2003. For men, risk of death over the years also reduced and was lower than that of females in the corresponding years with the exception of 2007 when it was higher than that for females.

Model IV has results with interaction term of slum of residence and education.

Mortality among residents of Viwandani with no education was about 18% higher than that of their Korogocho counterparts with no education. On the other hand having primary education among Viwandani residents lowers the risk of death by 29% while secondary or higher education lowered the risk by 48% compared with Korogocho residents with no education. Korogocho residents with secondary or higher education had about 22% lower mortality compared to those with no education in Korogocho.

Model V has results with interaction term of ethnicity and calendar year. Risk of death among the Kikuyu only substantially reduced in 2006 by 24% compared to the 2003 level while among the Luo, risk of death steadily decreased in all the years from 2.6 times higher risk to 12% higher risk compared with the Kikuyu in 2003. Among the other ethnicities, risk of death in the years after 2003 was lower but the decline was not steady as it was for the Luo.

Model VI has all the interaction terms. Results show that males of Korogocho had a 22% lower mortality compared with females in same slum while males in Viwandani had a 26% lower mortality compared with Korogocho females. Mortality among females over the years reduced and was higher than that for men in 2003, 2005 and 2006 and lower than that of men in 2007. For both slums, risk of death was lowest among individuals with secondary or higher education. The risk was lowest in Viwandani when compared to Korogocho residents with no formal education. Compared to the Kikuyu in 2003, risk of death was highest amongst the Luo as compared with all the other ethnicities.

I explored to find out whether age-specific mortality patterns varied by gender or area of residence. I used the piecewise exponential model to be able use the variable age group and its interaction with gender and slum of residence. Results showed that the

risk of death in the various age categories did not vary much by gender and slum (results not shown).

Table 11: Hazard ratios from Cox proportional hazard regression models with interaction terms

Variables	Model with no interactions (I) HR (95% CI)	Model with gender*slum interaction (II) HR (95% CI)	Model with gender* calendar year interaction (III) HR (95% CI)	Model with slum*education interaction (IV) HR (95% CI)	Model with ethnicity*calendar year interaction (V) HR (95% CI)	Full model with all 4 interactions (VI) HR (95% CI)
Gender						
Females	1.00	1.00	1.00	1.00	1.00	1.00
Males	0.87** (0.78,0.96)	0.99 (0.86,1.13)	0.70** (0.57,0.87)	0.86** (0.78,0.96)	0.87** (0.78,0.96)	0.78* (0.62,0.98)
Slum						
Korogocho	1.00	1.00	1.00	1.00	1.00	1.00
Viwandani	0.74*** (0.62,0.88)	0.90 (0.73,1.11)	0.74*** (0.62,0.88)	1.18 (0.80,1.75)	0.74*** (0.62,0.88)	1.32 (0.89,1.98)
Ethnicity						
Kikuyu	1.00	1.00	1.00	1.00	1.00	1.00
Luhya	0.83* (0.70,0.99)	0.83* (0.70,0.98)	0.83* (0.70,0.99)	0.83* (0.70,0.99)	0.97 (0.69,1.38)	0.99 (0.70,1.41)
Luo	1.77*** (1.55,2.03)	1.76*** (1.53,2.01)	1.77*** (1.55,2.03)	1.76*** (1.54,2.02)	2.59*** (2.00,3.34)	2.62*** (2.02,3.38)
Kamba	0.60*** (0.51,0.70)	0.60*** (0.51,0.71)	0.60*** (0.51,0.70)	0.60*** (0.51,0.71)	0.49*** (0.34,0.71)	0.51*** (0.36,0.74)
Cushites	0.61*** (0.47,0.80)	0.61*** (0.46,0.79)	0.61*** (0.47,0.80)	0.63*** (0.48,0.82)	0.34** (0.17,0.71)	0.35** (0.17,0.73)
Other	0.56*** (0.43,0.72)	0.56*** (0.43,0.73)	0.56*** (0.43,0.72)	0.57*** (0.44,0.74)	0.56* (0.32,0.98)	0.59 (0.34,1.03)
Wealth						
Poorest	1.00	1.00	1.00	1.00	1.00	1.00
Middle	0.88 (0.74,1.04)	0.87 (0.74,1.03)	0.88 (0.74,1.04)	0.87 (0.73,1.03)	0.87 (0.74,1.03)	0.86 (0.73,1.02)
Wealthiest	0.82** (0.71,0.93)	0.81** (0.71,0.93)	0.81** (0.71,0.93)	0.81** (0.71,0.93)	0.81** (0.71,0.93)	0.81** (0.71,0.92)
Unknown	1.98*** (1.37,2.85)	1.99*** (1.38,2.87)	1.97*** (1.37,2.84)	1.97*** (1.37,2.83)	1.97*** (1.36,2.83)	1.96*** (1.36,2.83)
Education						
No education	1.00	1.00	1.00	1.00	1.00	1.00
Primary	0.90 (0.74,1.09)	0.88 (0.72,1.07)	0.90 (0.74,1.10)	0.99 (0.80,1.24)	0.89 (0.73,1.09)	0.96 (0.77,1.20)
Secondary/higher	0.66*** (0.53,0.83)	0.65*** (0.52,0.82)	0.67*** (0.53,0.83)	0.78 (0.60,1.01)	0.66*** (0.53,0.83)	0.74* (0.57,0.97)
Unknown	0.94 (0.67,1.33)	0.94 (0.66,1.32)	0.95 (0.67,1.34)	0.94 (0.59,1.49)	0.93 (0.66,1.31)	0.92 (0.58,1.47)
Calendar year						
2003	1.00	1.00	1.00	1.00	1.00	1.00
2004	0.91 (0.78,1.06)	0.91 (0.78,1.06)	0.77* (0.61,0.98)	0.91 (0.78,1.06)	0.92 (0.71,1.19)	0.80 (0.59,1.09)
2005	0.78** (0.67,0.92)	0.78** (0.67,0.92)	0.74* (0.59,0.93)	0.79** (0.67,0.92)	0.94 (0.73,1.22)	0.88 (0.65,1.19)
2006	0.74*** (0.63,0.87)	0.74*** (0.63,0.87)	0.65*** (0.52,0.83)	0.74*** (0.63,0.87)	0.76* (0.58,1.00)	0.68* (0.50,0.93)
2007	0.78** (0.67,0.91)	0.78** (0.66,0.91)	0.59*** (0.46,0.75)	0.78** (0.67,0.91)	0.98 (0.76,1.26)	0.75 (0.55,1.01)



Gender*Slum				
Males#Viwandani	0.71**	(0.57,0.88)		0.72** (0.58,0.90)
Gender*Year				
Males#2004	1.33	(0.98,1.81)		1.32 (0.97,1.80)
Males#2005	1.12	(0.82,1.54)		1.16 (0.84,1.59)
Males#2006	1.25	(0.91,1.72)		1.25 (0.91,1.72)
Males#2007	1.62**	(1.18,2.22)		1.70** (1.23,2.34)
Slum*Education				
Viwandani#primary	0.61*	(0.41,0.90)		0.65* (0.44,0.97)
Viwandani#secondary	0.56**	(0.36,0.85)		0.62* (0.40,0.95)
Viwandani#unknown	0.76	(0.38,1.53)		0.81 (0.40,1.64)
Ethnicity*year				
Luhya#2004			1.10 (0.67,1.81)	1.07 (0.65,1.76)
Luhya#2005			0.76 (0.45,1.29)	0.75 (0.44,1.27)
Luhya#2006			0.82 (0.48,1.41)	0.80 (0.47,1.38)
Luhya#2007			0.63 (0.37,1.07)	0.60 (0.35,1.02)
Luo#2004			0.70 (0.48,1.02)	0.68* (0.46,1.00)
Luo#2005			0.55** (0.37,0.81)	0.54** (0.36,0.80)
Luo#2006			0.75 (0.51,1.12)	0.74 (0.50,1.10)
Luo#2007			0.44*** (0.29,0.66)	0.42*** (0.28,0.63)
Kamba#2004			1.52 (0.93,2.48)	1.47 (0.90,2.39)
Kamba#2005			1.04 (0.62,1.73)	1.01 (0.61,1.70)
Kamba#2006			1.48 (0.89,2.44)	1.44 (0.87,2.38)
Kamba#2007			1.15 (0.71,1.87)	1.08 (0.67,1.77)
Cushites#2004			1.59 (0.62,4.05)	1.57 (0.62,4.01)
Cushites#2005			2.45* (1.04,5.81)	2.43* (1.03,5.75)
Cushites#2006			1.69 (0.67,4.29)	1.70 (0.67,4.31)
Cushites#2007			2.15 (0.89,5.18)	2.12 (0.88,5.11)
Others#2004			1.27 (0.59,2.73)	1.22 (0.57,2.63)
Others#2005			0.67 (0.28,1.60)	0.65 (0.27,1.57)
Others#2006			1.29 (0.59,2.81)	1.26 (0.58,2.74)
Others#2007			0.82 (0.37,1.81)	0.76 (0.34,1.70)

Likelihood-ratio test LR chi2(8) =68.76; Prob > chi2<0.0001 (Assumption: smaller model nested in bigger model)

\* p<0.05, \*\* p<0.01, \*\*\* p<0.001

## 4.4 Discussion

Crude death rates are heavily influenced by the underlying age structure of the population and thus not suitable for making comparisons across populations. In this analysis, however, a few clear patterns stood out and remained in line with the adjusted results. Mortality among females was generally higher than males. This result was unexpected as most studies show the reverse. Also, while the slums appear similar in many aspects, Korogocho had a markedly higher crude death rate than Viwandani. Controlling for age using the WHO global standard and the INDEPTH standard showed that mortality among women remained higher than that among men. This is in contrast to the national trend that show that adult mortality is generally higher among men than women except for the age bracket 20-29 year <sup>[13]</sup>.

The huge difference in mortality rates by slum seen among women greatly diminished upon controlling for age. However, among men, a big difference by slum of residence remained. This implies that the age structure of women in Viwandani slum was such that those age groups of women with a tendency to have higher mortality were underrepresented in the population. Indeed from the population pyramid for Viwandani (Figure 4(b)), it can be seen that there is a predominance of men in the age categories 25-49 years and few women especially after the age 60 and above. The observation that the standardised rates by both standards are higher than the observed crude rates means that the population of the slums is over represented by age groups which normally have lower mortality experience. That is to say that, if the observed age-specific mortality rates were applied to the slum population with an age structure similar to that of the WHO global standard or the INDEPTH standard, the mortality rates would have been higher than those observed.

Life expectancy at the age of 15 years among slum residents is low at 41 years for females and 44 years for males. Compared with results from other health and demographic surveillance sites, with the exception of Bandim in Guinea-Bissau and Manhica in Mozambique (females), life expectancy at the age of 15 at the NUHDSS site is generally low. Also, it emerges clearly that Nairobi site is the only site where the life expectancy at 15 for males is higher than that for females <sup>[55]</sup>.

The probability of a 15-year old dying before reaching his/her 60<sup>th</sup> birthday was high, estimated at 558 per 1000 among women and 476 per 1000 among men. The observed probabilities of death between 15 and 60 years in this population are generally high compared with other published data on sub-Saharan Africa. Data from the INDEPTH publication, *Population and Health in Developing Countries* shows that in all the 19 sites that contributed to that publication, with the exception of the Rufiji and Dar es Salaam DSS sites, the probability of dying between the age of 15 and 60 was higher in males than females <sup>[55]</sup>. It should be noted, however, that the age bracket considered in defining adult mortality in the INDEPTH publication referred to above was 20-50 years not the conventionally and commonly used age bracket of 15-60 years <sup>[55]</sup>. In this study, before the age of 25 years, mortality was higher among males. However, from that age onward it became higher among females and only converged at a much later age of about 65 years. While the switch at the age of 25 years might be attributed to excess mortality due to HIV/AIDS among females, it is not clear why mortality persistently remains higher among females beyond the age of 49 years. However, while HIV prevalence in most other settings in sub-Saharan Africa peaks in the 30s and 40s for females and males respectively, by contrast, results from the two slums show that HIV prevalence was highest in the oldest age groups studied 50 to 54 years of age among males and 45 to 49 years among females <sup>[234]</sup>. This implies that more HIV/AIDS associated mortality should be expected in older age groups as these results suggest.

The risk of death was higher in Korogocho than Viwandani slum. However, gender differences in the risk of death were only observed in Viwandani slum. While the two slums are similar in many ways, they are different in some respects that might explain the current results. Viwandani slum is located near an industrial estate and thus is home to most young people who work in the industries. Earlier research by APHRC showed that residents of Viwandani were generally young adults, many of whom live on their own, have better educational attainment and better income than their Korogocho counterparts <sup>[235]</sup>. To what extent these differences explain the observed difference in risk of death remains unclear.

Marked differences in the risk of death were also observed by ethnicity. Ethnicity has been reported in many studies to be associated with health status in Kenya including

child survival and HIV status <sup>[12, 13, 236]</sup>. Findings in this study showed that the Luo ethnic group had the highest risk of death. Previous HIV prevalence studies have shown that HIV prevalence in Kenya is highest among the Luo ethnic group <sup>[12, 13]</sup>. Why this is the case is not fully understood, but it may be linked to cultural practices that increase susceptibility to HIV infection, including wife inheritance and lack of male circumcision <sup>[13, 237]</sup>. Given this background, it is likely that the excess mortality seen among the Luo is attributable to HIV/AIDS. It is also important to note that, apart from the Luo, all other ethnic groups have a lower risk of death than the Kikuyu. The Kikuyu ethnic group come from the Central region of the country neighbouring the city. In recent years many Kikuyu youths have been involved with an illegal cult-like organisation which promotes violence, extortion and other forms of criminal activities. In the process, its members suffered deaths meted out by community members and the law enforcement agencies <sup>[238]</sup>. This might explain the high mortality especially among males in this ethnic group, which has lower HIV prevalence than most other ethnicities.

The finding that the wealthiest and those with secondary or higher education had lower risk of death than the poorest and those with no formal education respectively might be related to a lower risk of exposure to HIV and higher access to health care among those infected. An earlier study in the two slums showed that those with no formal schooling had the highest prevalence of HIV but no difference by wealth status <sup>[234]</sup>. Previous research reports have found an association between higher education, wealth and HIV status, and thus one would have expected to see higher risk of death from those categories. However, even if wealth and education were related to a higher risk of infection with HIV nationally, the situation in the slums might be different. The poorest might be involved in more risk-taking behaviour such as drug abuse and high-risk commercial sex work <sup>[37]</sup>. Access to and utilisation of health care services might also be influenced by the two factors.

The trend of a decreasing risk of death over the years observed in the results may be a true reflection of mortality trends in this population but there could be other explanations. If mortality is decreasing, this could be attributed to recent improvements in the management of HIV/AIDS and associated opportunistic infections as has been reported in other studies <sup>[88, 239, 240, 241, 242]</sup>. Anti-retroviral therapy not only improves

quality of life of infected individuals, it also prolongs life by more than 10 years in most cases. There has been marked increase in access to antiretroviral therapy since the early 2000s through financial support from the United States government <sup>[243]</sup>. There have also been efforts to improve maternal health in order to achieve the fifth MDG; however due to lack of appropriate monitoring, the impact these efforts might have had on maternal health and mortality is uncertain.

Among women, the wealthiest had no better survival than the poorest, while among men the wealthiest had a significantly lower risk of death. Also, in Viwandani slum, there was no survival advantage of the wealthiest while in Korogocho a survival advantage of the wealthiest was evident. Poorer males might be involved in more risky jobs or other activities such as robbery and hence result into increased risk of death from injuries. The downward trend in mortality over the years was more marked among residents of Korogocho and females, which is consistent with this being related to HIV/AIDS as a cause of death. HIV infection is generally more prevalent in Korogocho slum and among females. With ongoing rollout of ARVs in Kenya, improvements in adult survival are expected <sup>[244]</sup>. Thus, it should also be expected for mortality decline to be more marked in populations where the burden of HIV/AIDS is highest. The downward trend in risk of death among females and absence of a similar trend among males may be a pointer to differences in the drivers of mortality between the two sexes. While HIV/AIDS is an important cause of death among men, death from injuries are also important and, since there are no interventions targeted at injuries, no trend would be expected in a short period of time.

## **4.5 Conclusions**

Adult mortality in the slums is high and the life expectancy of women and men of these slums is low compared with several other populations under surveillance in sub-Saharan Africa. There were mortality differentials by slum of residence and gender. As has been reported in some other populations affected by HIV/AIDS, adult mortality was higher among women, contrary to expectation and historical observations in many populations. The difference in mortality by slum is unaccounted for by demographic differences in the two slums. The decline in mortality seems to be real, particularly for women, and is most likely attributable to reduction in HIV/AIDS related deaths.

## **5. Cause-specific adult mortality**

### **5.1 Introduction**

While data on all-cause adult mortality in Kenya and sub-Saharan Africa region as a whole are scanty, the situation for specific causes of death and their relative contribution to the overall burden of adult deaths at a population level is worse. In Kenya, many deaths occur outside of a health facility and, as such, no autopsy is carried out to ascertain cause of death. Reporting of death is a legal requirement to allow burial to take place in Kenya and currently about 50% of deaths are reported <sup>[9]</sup>. However, most of these deaths are not assigned causes. Even for deaths that take place in health facilities, the cause of death is not often certified and recorded in standard format, for example using ICD 10.

In recent years there has been emphasis on strengthening the health information system in Kenya and certain aspects of the system do report well but cause-of-death data are not reported. The urgent need for cause-of-death data in many developing countries has led researchers to use alternative approaches to get these data. Community-based social autopsies, often referred to as verbal autopsy, have been used to fill the data gap, the weaknesses of the approach notwithstanding <sup>[69, 72, 75, 245, 246]</sup>. In the absence of vital registration and physician-certified death records, verbal autopsies have been found to be the next best option in arriving at probable causes-of-death for public health use. The verbal autopsy tool has been found to have variable sensitivities and specificities depending on the disease and methods employed in ascertaining cause of death <sup>[76, 245]</sup>. Overall, these have been acceptable; consequently cause-of-death data from verbal autopsy have been widely published and used.

### **Causes of death**

While current data indicate that HIV/AIDS is a leading cause of adult death in most of sub-Saharan Africa, other previously less important disease entities are emerging <sup>[1, 61, 89, 247, 248]</sup>. The epidemiologic transition theory posits that with social development, improvements in health care, nutrition and general living standards of populations, the

epidemiology of diseases shifts from a predominance of infectious and under-nutrition disorders to that of non-communicable and diseases of life style <sup>[63]</sup>. This has been clearly observed in established market and emerging economies. The epidemiological picture for most of sub-Saharan Africa is increasingly becoming mixed. Old and new infectious diseases such as tuberculosis and HIV/AIDS remain major causes of ill-health and death while at the same time non-communicable diseases such as diabetes mellitus and cancer are increasingly becoming major causes of death <sup>[249]</sup>. The surge in diseases of lifestyle mirrors increasing prevalence of known risk factors for non-communicable diseases such as hypertension, obesity, smoking and alcoholism among others <sup>[250]</sup>.

### **Slum Context**

By the very nature of slums, it is expected that certain causes of ill-health and their risk factors are likely to be different from those of other parts of urban and rural populations. In Kenya, HIV/AIDS is a leading cause of adult mortality. HIV prevalence in the informal settlements is higher than that urban areas and Kenya as a whole and this probably translates into higher mortality among residents attributable to HIV/AIDS <sup>[1, 234]</sup>. By gender, HIV prevalence both for the slums and Kenya as a whole is highest among females which may lead to higher mortality among females attributable to HIV/AIDS and to higher overall female adult mortality, although in most populations it is lower than that of men. In populations with high HIV prevalence, tuberculosis closely follows HIV/AIDS as a cause of death, it being an opportunistic infection among HIV infected individuals <sup>[251]</sup>. The poor housing and crowding in slums means that the population is at a heightened risk of tuberculosis transmission. With regard to injuries as a cause of death, the unplanned housing infrastructure and illegal powers connections exposes residents to a higher risk of fires, electrocution and other forms of injury. Recent research indicates that injuries account for a sizeable proportion of cause of adult death <sup>[1]</sup>. While slums are thought to be a hotbed for infectious diseases and injury, non-communicable diseases particularly hypertension and diabetes mellitus, are also emerging as important <sup>[252, 253]</sup>.

While knowledge of the extent of the burden attributable to various causes of death is important in planning interventions including allocation of resources, it is additionally

useful in anticipating the likely impact on families and the population as this may vary by cause of death. Knowing the major causes of deaths focuses research, including identification of risk factors, coverage of interventions, and use of health care services. This chapter explores causes of adult death and provides comparisons in terms of mortality rates but also factors associated with risk of death by cause.

## **5.2 Methods and data sources**

### **Cause of death classification**

Codes for cause of death data were linked to the corresponding disease entities. Where the number of cases for a given disease category was small, they were collapsed into bigger categories for analysis purposes. About 9% of all deaths had an indeterminate cause, that is, the physicians reviewing the verbal autopsies failed to establish a probable cause of death. Another 20% of all deaths had no information on the cause of death because no verbal autopsy interview was done. Absence from home, change of residence or out-migration of credible respondents are some of the reasons for missing cause of death information but refusals to consent to give the interview also contribute to this. For computing cause-specific mortality rates, the cases with indeterminate and missing cause of death data were proportionately redistributed to the known causes stratified on age category and gender. Further assessment of how deaths with cause of death information compare with those without cause of death information was carried out using logistic regression controlling for age at death, year the death occurred, slum of residence, ethnicity, wealth status, education level, household relations and mobility. Mobility was constructed as an index taking into account number of out- and in-migration episodes and change of residence within the slum per unit time (duration). Internal movements (change of residence) episodes were given a weight of 1 while external movement (out- and in-migration) were given a weight of 2.

### **Modelling cause-specific hazard ratios**

To be able to assess risk of death due to specific cause of death, the failure event was redefined as death of an adult from a specific cause or group of causes (and was assigned a value of 1) while the rest were considered censored or lost to follow up (assigned a value of zero) even if they died but of other causes.



### **Imputation for missing cause of death data**

Altogether approximately 29% (443) of the deaths reported did not have cause of death data due to missing (uncollected) data (69%) or inability of physicians to agree on a given cause of death (31%). In the real world, missing data may not be random and if this were the case, estimates from the realised sample may be biased depending on how strongly selective the missing information is and whether it is related to main outcome of interest.

Against this background, I explored missing information for cause of death using a set of variables on each of the individuals using multivariate methods that use multiple imputations with chained equations as described by van Buuren et al. <sup>[254]</sup> and implemented in Stata statistical software by user written programmes *ice* and *mim* <sup>[255]</sup> <sup>[256]</sup>. *Ice* does the multiple imputations (normally 5 to 10 are recommended) and *mim* carries out parameter estimations on the imputed data sets and in the process the estimates from the multiple datasets are combined according to Rubin rules <sup>[257]</sup>. The imputation process works on the assumption that the data are missing at random, meaning that the observed data can be used to predict the missing data. This assumption may not always hold true in real life situations.

Using *ice*, I imputed 5 multiple data sets using the following variables: gender, ethnicity, slum of residence, education attainment, wealth, calendar year since observation started and survival time. I used Cox proportional hazard models regression in the *mim* programme to analyse the stacked data sets and estimate the hazard ratios for each of the major cause-of-death categories. As recommended by van Buuren, survival time was included in the imputation equation after a logarithm transformation.

## **5.3 Results**

### **Causes of death**

Table 12 shows the proportions contributed by the different causes of death among adults who died between 2003 and 2007. HIV/AIDS had the highest percentage (34.4%), followed by injuries (21.5%) and tuberculosis (10.8%). In addition to injuries, non-communicable causes of death such as cancers, cardiovascular diseases, maternal

causes and other non-communicable diseases are important causes of death (CoD) accounting for over 25% of all causes of death.

Table 12: Causes of death in Korogocho and Viwandani among adults

Cause of death	Number	Percentage (including those with no CoD data)	Percentage (excluding those with no CoD data)
HIV/AIDS	368	24.3	34.4
Tuberculosis	116	7.7	10.8
Maternal causes	27	1.8	2.5
Injuries	230	15.2	21.5
Cancers	59	3.9	5.5
Renal diseases	24	1.6	2.2
CVD disorders	40	2.6	3.7
Other CNS disorders	24	1.6	2.2
Diabetes	23	1.5	2.1
Gastro-intestinal disorders	39	2.6	3.6
Meningitis	36	2.4	3.4
Pneumonia	30	2.0	2.8
Other communicable diseases	28	1.9	2.6
Other non-communicable diseases	26	1.7	2.4
Missing information/No verbal autopsy	306	20.2	
Indeterminate cause of death	137	9.1	
Total	1513	100.0	100.0

While the information in Figure 8 below is similar to that in Table 12, in the figure, the causes of death were restricted to 10 categories- less important causes were collapsed into “other communicable diseases” and “other non-communicable diseases”. The figure highlights the predominance of communicable diseases as leading causes of death in the two slums. Part of the gastrointestinal (GIT) causes in Table 12 above were redistributed to either “other communicable-mainly acute abdominal conditions associated with a fever” or other non-communicable diseases-mainly chronic liver conditions.

Because individuals without cause of death may be systematically different in important ways from those with cause of death data, I explored further how these two categories compare by key socio-demographic characteristics as shown in Table 13. If they are systematically different then estimates obtained from the observed data could be biased. Results in Table 13 show that from the age of about 75 years and above, deaths are less likely to have cause of death data than those aged 15-19 years. This point will be explored further later in Figure 9 below.

Figure 8: Percent distribution of cause of death grouped into top ten categories.

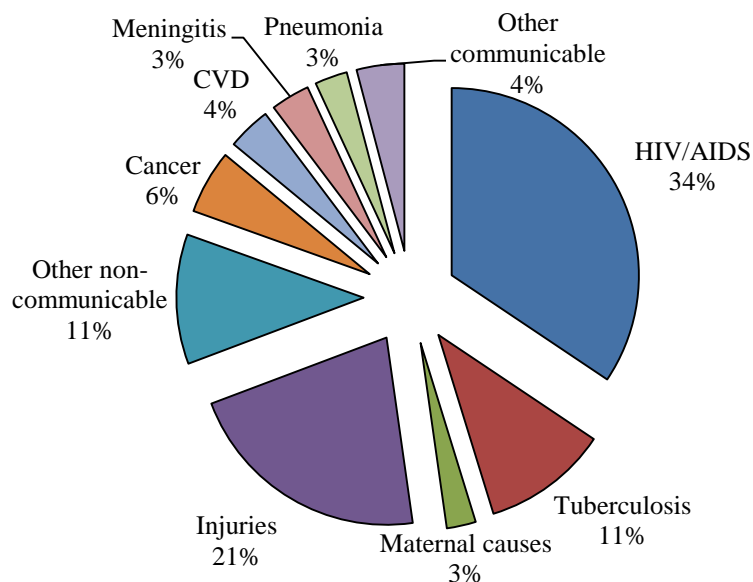


Table 13: Factors associated with having cause of death data from a logistic regression model

Variables	Univariate analysis			Multivariate model		
	Odds Ratio	(95% CI)	P value	Odds Ratio	(95% CI)	P value
Age group						
15-19yrs	1.00			1.00		
20-24yrs	0.88	(0.43;1.80)	0.719	1.05	(0.48;2.28)	0.899
25-29yrs	0.58	(0.30;1.14)	0.114	0.67	(0.32;1.42)	0.299
30-34yrs	0.56	(0.29;1.08)	0.083	0.69	(0.33;1.44)	0.318
35-39yrs	0.57	(0.29;1.13)	0.106	0.64	(0.29;1.37)	0.247
40-44yrs	0.59	(0.30;1.18)	0.134	0.68	(0.31;1.47)	0.322
45-49yrs	0.79	(0.37;1.68)	0.540	0.85	(0.36;1.97)	0.703
50-54yrs	0.59	(0.28;1.26)	0.177	0.65	(0.28;1.51)	0.319
55-59yrs	0.78	(0.34;1.82)	0.564	0.84	(0.33;2.15)	0.719
60-64yrs	1.00	(0.36;2.81)	0.996	1.20	(0.39;3.71)	0.746
65-69yrs	1.04	(0.35;3.07)	0.943	1.03	(0.32;3.38)	0.958
70-74yrs	0.85	(0.30;2.42)	0.767	0.96	(0.31;3.02)	0.944
75-79yrs	0.26	(0.08;0.87)	0.029	0.23	(0.06;0.88)	0.033
80-84yrs	0.29	(0.09;0.91)	0.033	0.25	(0.07;0.87)	0.030
85+yrs	0.24	(0.09;0.64)	0.004	0.27	(0.09;0.83)	0.022
Gender						
Females	1.00			1.00		
Males	1.06	(0.85;1.33)	0.607	1.03	(0.78;1.37)	0.814
Slum						
Korogocho	1.00			1.00		
Viwandani	0.71	(0.57;0.89)	0.003	0.68	(0.45;1.04)	0.073
Ethnicity						
Kikuyu	1.00			1.00		

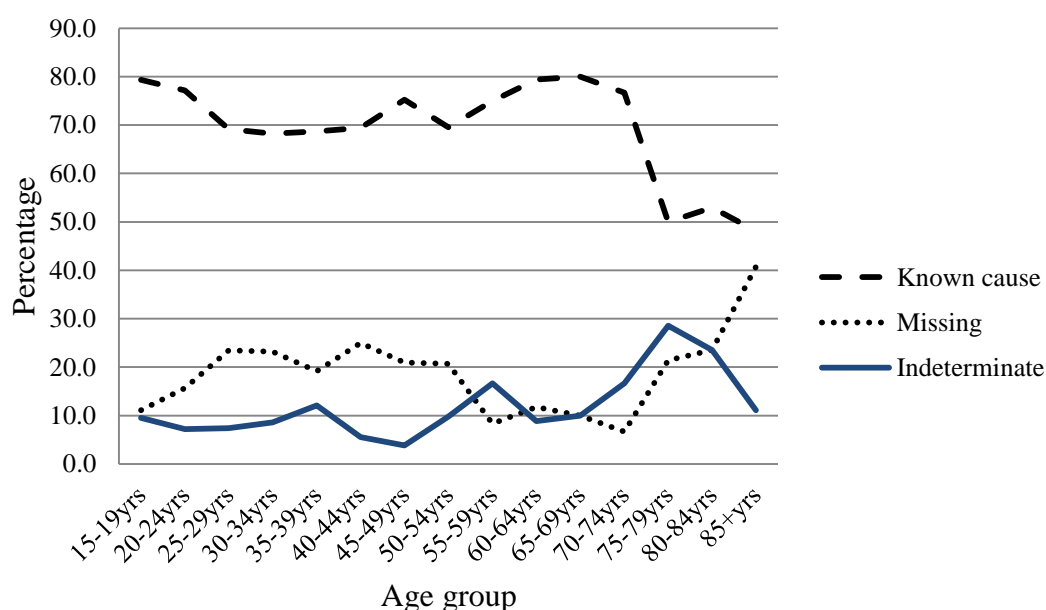
Luhya	1.14	(0.79;1.65)	0.493	1.08	(0.72;1.62)	0.693
Luo	1.36	(1.02;1.81)	0.037	1.26	(0.91;1.76)	0.161
Kamba	0.93	(0.67;1.30)	0.683	1.01	(0.69;1.48)	0.948
Cushites	1.20	(0.69;2.10)	0.514	1.45	(0.77;2.75)	0.251
Others	0.80	(0.47;1.36)	0.418	0.84	(0.47;1.49)	0.546
Wealth						
Poorest	1.00			1.00		
Middle	0.58	(0.43;0.78)	<0.001	0.74	(0.50;1.08)	0.121
Wealthiest	0.79	(0.60;1.03)	0.078	0.59	(0.42;0.82)	0.002
Unknown	1.72	(0.65;4.57)	0.278	1.08	(0.39;3.00)	0.888
Education						
No education	1.00			1.00		
Primary	0.93	(0.65;1.33)	0.707	0.80	(0.50;1.28)	0.357
Secondary/higher	0.84	(0.57;1.25)	0.395	0.81	(0.48;1.36)	0.424
Unknown	1.45	(0.65;3.25)	0.362	1.45	(0.60;3.51)	0.406
Year of death						
2003	1.00			1.00		
2004	1.19	(0.79;1.78)	0.400	1.13	(0.74;1.71)	0.572
2005	0.67	(0.46;0.97)	0.037	0.66	(0.44;0.97)	0.036
2006	0.33	(0.23;0.48)	<0.001	0.32	(0.22;0.46)	<0.001
2007	0.24	(0.17;0.35)	<0.001	0.23	(0.16;0.34)	<0.001
Mobility						
Not highly mobile	1.00			1.00		
Highly mobile	0.94	(0.69;1.28)	0.698	1.02	(0.73;1.43)	0.898
HH relations						
HH head	1.00			1.00		
Spouse	0.82	(0.62;1.10)	0.187	0.78	(0.55;1.13)	0.193
Child	1.93	(1.13;3.32)	0.017	1.69	(0.91;3.15)	0.097
Other	1.24	(0.82;1.88)	0.315	1.07	(0.66;1.72)	0.780

HH=Household

Residents of Viwandani were less likely to have a known cause of death compared with those of Korogocho but this became insignificant upon controlling for other covariates. Gender was not associated with risk of not having cause of death data. Individuals from the Luo ethnic group were about 36% more likely to have a known CoD compared with the Kikuyu but this was not significant when other factors were controlled for. The wealthiest individuals were significantly less likely to have a known CoD. From 2005, there seem to be a significant trend whereby the likelihood of a deceased person having a known CoD decreased with time. Overall individuals with no CoD data and those with CoD data do not seem to greatly differ along many characteristics with the exception of wealth, older age and the year the death occurred (2005-2007).

Figure 9 shows that the proportion of deaths with missing CoD data and those with indeterminate results sharply raise after the age of 70. This may mean that most of the unknown CoD among the elderly are mainly due to missing VA data but also due to indeterminate results seen to rise from about 70 years as compared with younger age groups.

Figure 9: Proportion of individuals with known cause of death, indeterminate and missing data by age.



### Age-adjusted cause-specific mortality rates

Table 14 shows the age adjusted cause-specific mortality rate using the two standards by gender and Table 15 shows the ranking of the top 10 causes of death based on age adjusted cause-specific mortality rates. The adjusted cause-specific mortality rates using both standards were higher than the crude cause-specific death rates. Generally, the adjusted cause-specific death rates using the WHO global standard were higher than those adjusted using the INDEPTH standard.

The age-adjusted death rate due to HIV/AIDS among females was about double that among males irrespective of the standard used for adjustment. The age-adjusted death rates due to tuberculosis in both sexes were very similar while that due to injuries was over 6 times higher among males compared with females. The cancer mortality rate after adjusting for age was more than twice as high in females as in males. Mortality rate due to diabetes among women was more than 5 times (after adjusting for age using WHO standard) and about 4.5 times (after adjusted for age using INDEPTH standard) higher among females than males. Mortality rates due to pneumonia were higher among females compares to males by over 3.6 times as per estimates obtained after adjusting for age using WHO standard and by 4.5 times as per estimates after adjusting using the INDEPTH standard.

Table 14: Crude and age adjusted cause-specific mortality rates per 100,000 person-years

Cause of death by sex	Person years	CDR	Adjusted by WHO standard		Adjusted by INDEPTH standard	
			ASCDR	(95% CI)	ASCDR	(95% CI)
HIV/AIDS						
Female	78166	269	338	(276;400)	307	(254;360)
Male	114305	138	176	(135;216)	155	(121;189)
Total	192471	191	229	(196;262)	207	(179;235)
Tuberculosis						
Female	78166	59	91	(54;128)	80	(49;112)
Male	114305	61	99	(60;138)	85	(53;116)
Total	192471	60	91	(66;116)	80	(59;100)
Maternal						
Females	73793	37	31	(18;44)	31	(19;43)
Injury						
Female	78166	28	34	(12;55)	32	(13;51)
Male	114305	182	202	(162;243)	201	(166;236)
Total	192471	120	135	(110;161)	131	(109;154)
Cancer						
Female	78166	47	155	(92;217)	125	(74;175)
Male	114305	19	75	(31;120)	56	(24;88)
Total	192471	31	101	(66;136)	78	(52;105)
Renal diseases						
Female	78166	12	20	(0;40)	17	(0;33)
Male	114305	13	47	(13;82)	40	(13;68)
Total	192471	13	35	(14;56)	30	(13;47)
Cardiovascular diseases						
Female	78166	27	102	(49;155)	83	(41;125)
Male	114305	17	90	(40;140)	68	(31;105)
Total	192471	21	91	(56;126)	72	(45;100)
Central nervous disorders						
Female	78166	12	37	(6;67)	27	(4;51)
Male	114305	13	23	(6;41)	21	(5;36)
Total	192471	13	31	(13;48)	24	(11;37)
Diabetes						
Female	78166	19	120	(57;183)	91	(42;141)
Male	114305	7	27	(2;51)	23	(2;44)
Total	192471	12	69	(37;101)	52	(28;77)
Gastrointestinal diseases						
Female	78166	18	36	(8;64)	31	(7;54)
Male	114305	22	43	(13;73)	35	(12;58)
Total	192471	20	37	(19;56)	31	(17;46)
Meningitis						
Female	78166	23	23	(10;37)	22	(10;34)
Male	114305	16	18	(8;28)	16	(7;25)
Total	192471	19	20	(12;28)	19	(12;26)
Pneumonia						
Female	78166	12	21	(3;38)	12	(4;20)
Male	114305	18	74	(27;121)	50	(20;80)
Total	192471	16	47	(22;71)	31	(15;47)
Other infectious diseases						
Female	78166	13	21	(0;43)	15	(0;30)
Male	114305	16	26	(5;47)	18	(8;27)
Total	192471	15	25	(10;40)	18	(8;27)
Other NCD						
Female	78166	14	38	(8;69)	31	(7;55)
Male	114305	13	49	(16;83)	40	(14;67)
Total	192471	14	45	(22;68)	37	(18;55)

*NCD Non-communicable diseases; CDR Unadjusted cause-specific death rate; ASCDR Age standardised cause-specific death rate.*

Overall, HIV/AIDS is the leading cause of death followed by injuries (Table 15).

Among females, the leading cause is HIV/AIDS followed by cancer and diabetes while for males the leading causes were; injuries, HIV/AIDS, tuberculosis and cardiovascular diseases in that order. Although there are some differences in the ordering of importance of cause of death by type of standard used for adjustment, most causes of death were the same in the list of top 10 causes of death. It is also important to note that among women, injuries made it to the top 10 causes of death, coming before maternal causes of death.

Table 15: Top 10 leading causes of death in Korogocho and Viwandani by sex

Rank	Females & males		Females		Males	
	Cause-specific mortality rate adjusted using WHO standard	Cause-specific mortality rate adjusted using INDEPTH standard	Cause-specific mortality rate adjusted using WHO standard	Cause-specific mortality rate adjusted using INDEPTH standard	Cause-specific mortality rate adjusted using WHO standard	Cause-specific mortality rate adjusted using INDEPTH standard
1	HIV/AIDS	HIV/AIDS	HIV/AIDS	HIV/AIDS	Injury	Injury
2	Injury	Injury	Cancer	Cancer	HIV/AIDS	HIV/AIDS
3	Cancer	Tuberculosis	Diabetes mellitus	Diabetes mellitus	Tuberculosis	Tuberculosis
4	Tuberculosis	Cancer	Cardiovascular diseases	Cardiovascular diseases	Cardiovascular diseases	Cardiovascular diseases
5	Cardiovascular diseases	Cardiovascular diseases	Tuberculosis	Tuberculosis	Cancer	Cancer
6	Diabetes mellitus	Diabetes mellitus	Other NCD	Injury	Pneumonia	Pneumonia
7	Pneumonia	Other NCD	Central nervous disorders	Maternal causes*	Other NCD	Renal diseases*
8	Other NCD	Pneumonia*	GIT disorders	GIT disorders*	Renal diseases	Other NCD*
9	GIT disorders	Maternal causes*	Injury	Other NCD*	GIT disorders	GIT disorders
10	Renal disorders	GIT disorders*	Maternal causes	Central nervous disorders	Diabetes mellitus	Diabetes mellitus

*NCD= Non communicable diseases; GIT= Gastro-intestinal disorders; \* same rank within category; Other NCD-Non-communicable diseases other than cancer, injury, maternal causes, diabetes, renal and cardiovascular disorders.*



### **Modelling mortality by cause of death**

Table 16 shows hazard ratios from several regression models based on cause-specific death rates from the main causes of death as outlined earlier. Hazard ratios were also computed for broader categories that make sense from a public health intervention perspective such as HIV/AIDS and tuberculosis, non-HIV causes and so forth.

#### **HIV/AIDS:**

The results show that the risk of death due to HIV/AIDS was about 55% lower among males than females after controlling for residence, ethnicity, wealth status, educational attainment and calendar year. The risk of death due to HIV/AIDS was also lower among residents of Viwandani by about 47%. The Luo ethnic group was more than 3 times as likely to die of HIV/AIDS compared with the Kikuyu, while the Kamba were 38% less likely to die of HIV/AIDS compared with the Kikuyu. The results also show a consistent trend of reduction in risk of death due to HIV/AIDS over the years, from a reduction of 28% in 2004 to 78% in 2007. Wealth status and educational attainment levels were not significantly associated with risk of death from HIV/AIDS.

#### **Tuberculosis:**

Results show that residents of Viwandani were 50% less likely to die of tuberculosis than those of Korogocho. However, there were no significant differences in mortality due to tuberculosis by gender and education levels. The Luo ethnic group had 73% excess risk of dying of tuberculosis compared with the Kikuyu. Individuals whose wealth status was not known were nearly 4 times as likely to die of TB compared with the poorest while the wealthiest were 40% less likely to die from tuberculosis as compared with the poorest. Significant reduction (62%) in mortality due to TB was seen in 2007 but not in the other years though the general trend shows reduction in mortality since 2005 although insignificant.

#### **HIV/AIDS or Tuberculosis**

Since many times HIV/AIDS and tuberculosis infections do occur together or may not be easily distinguishable, I combined death due to HIV/AIDS with those from tuberculosis to estimate the risk of death from either or both. Results show that among

males, mortality due to HIV/AIDS or tuberculosis was about 46% lower than that observed among females. Residents of Viwandani had a 47% lower risk of death from either HIV/AIDS or tuberculosis compared with residents of Korogocho. The Luo ethnic group were about 3 times as likely to die from either HIV/AIDS or tuberculosis compared with the Kikuyu while the Kamba were 32% less likely to die of HIV/AIDS or tuberculosis. The wealthiest were 24% less likely to die of HIV/AIDS or TB compared with the poorest. Since 2003, the risk of death due to HIV/AIDS or tuberculosis has consistently reduced to a maximum of 75% in 2007 compared to the levels in 2003.

### **Communicable diseases other than HIV/AIDS and tuberculosis**

This category consists mainly of infectious diseases such as meningitis, pneumonia, malaria and other febrile illnesses. Gender, slum of residence, educational attainment and wealth status were not associated with risk of death due to diseases in this category. However by ethnicity, the Luo ethnic group had more than two times the risk of death from diseases in this category compared with the Kikuyu. Although there seem to be a downward trend in risk of death from diseases in this category since 2004, this was not statistically different from the 2003 rates.

### **All communicable diseases**

This category includes all communicable diseases such as HIV/AIDS, tuberculosis, malaria, meningitis and pneumonia among others. From Table 16 it can be seen that men had 38% lower risk of dying of any communicable disease than women while residents of Viwandani had a 47% lower risk. Luo ethnic group had close to three times the risk of dying of communicable diseases than Kikuyu while the Kamba had about 34% lower risk of dying from communicable disease than the Kikuyu. Individuals ranked as the wealthiest in the two communities were about 24% less likely to die from a communicable disease. From 2005, risk of death from a communicable disease significantly reduced by 34% in 2005, 58% in 2006 and 68% in 2007.

### **Injury/external causes of death**

The risk of death from injuries was about 7.4 times higher among men than women (Table 16). There was no significant difference by slum of residence and educational level attainment. The Luhya ethnic group had 49% lower risk of death from injuries

than the Kikuyu. Although not statistically significant the Luo also had lower mortality from injuries compared with the Kikuyu. The Kamba had significantly lower risk of death from injuries; 60% lower than the Kikuyu. The risk of death from injuries decreased with increase in wealth status with the wealthiest having 53% lower risk compared with the poorest. Individuals whose wealth status was not known had about 2.8 times higher risk of death from injuries as did the poorest. There was no significant difference in risk of death from injuries by calendar year from 2003 to 2007. Compared with women, men's risk of death from intentional injuries was higher than that from unintentional injuries. The risk of death from intentional injuries increased significantly from 2005 to 2007.

### **Cancer**

Results in Table 16 show that men had 64% lower risk of death from all cancers than women. There were no significant differences in risk of death from cancer by ethnicity, wealth status and educational attainment.

### **Non-Communicable diseases other than injuries and cancers**

This category is mainly composed of cardiovascular diseases, maternal causes, renal diseases, central nervous disorders and diabetes. Men had about 46% lower risk of dying from a disease in this category than women. Slum of residence, ethnicity wealth status and educational attainment were all not associated with risk of dying from diseases in this category.

Table 16: Hazard ratios from Cox proportional hazard regression models by cause of death (cause-specific rates)

Variable	Hazard ratios												
	Communicable diseases					Non-communicable diseases							
	HIV/AIDS	Tuberculosis	HIV/TB	Other CD <sup>s</sup>	All CD	All injuries	Un-intentional	Intentional	Cancer	Other NCD <sup>t</sup>	All NCD	Non-HIV	Missing CoD
Gender													
Females	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Males	0.45***	0.96	0.54***	1.18	0.62***	7.38***	6.41***	9.26***	0.36***	0.54***	1.45***	1.30**	0.81*
Slum													
Korogocho	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Viwandani	0.53**	0.50*	0.53***	0.57	0.53***	0.73	0.82	0.63	1.18	0.90	0.83	0.72**	0.99
Ethnicity													
Kikuyu	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Luhya	1.15	1.07	1.13	0.96	1.10	0.51**	0.48*	0.58	1.05	0.89	0.65**	0.75*	0.76
Luo	3.45***	1.73*	2.99***	2.29**	2.87***	0.80	0.80	0.82	1.86	1.23	0.97	1.25*	1.50**
Kamba	0.62*	0.83	0.68*	0.56	0.66**	0.40***	0.48**	0.26***	1.29	0.69	0.55***	0.58***	0.59***
Cushites	0.30**	1.63	0.63	1.50	0.82	0.33*	0.20*	0.52	0.31	0.90	0.49**	0.74	0.57*
Others	0.63	0.25	0.53*	0.30	0.49**	0.48**	0.62	0.26*	1.09	0.51	0.53**	0.47***	0.63*
Wealth													
Poorest	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Middle	1.18	0.78	1.06	0.78	1.01	0.63*	0.68	0.52	0.46	0.73	0.64**	0.68**	1.06
Wealthiest	0.83	0.60*	0.76*	0.77	0.76*	0.47***	0.58*	0.33***	1.08	0.82	0.66***	0.67***	1.16
Unknown	1.08	3.84**	1.78	1.80	1.79	2.76**	3.75***	1.21	1.49	1.87	2.56***	2.66***	1.34
Education													
No education	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Primary	0.84	1.08	0.90	1.41	0.99	0.79	1.41	0.31*	0.56	1.03	0.71*	0.83	1.08

Secondary & higher	0.67	0.72	0.69	0.70	0.70	0.58	1.03	0.24**	0.46	0.95	0.57**	0.60**	0.81
Unknown	1.17	1.41	1.22	1.28	1.25	1.19	2.49	0.23	1.94	0.28	0.87	0.97	0.74
Calendar year													
2003	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
2004	0.72*	1.19	0.81	1.20	0.86	1.27	1.47	0.17	0.60	0.95	1.02	1.08	0.82
2005	0.61***	0.73	0.63***	0.80	0.66***	0.90	0.75	1.83	0.59	0.79	0.80	0.79*	1.10
2006	0.31***	0.62	0.37***	0.75	0.42***	1.04	0.49*	4.39**	0.51	0.59*	0.75	0.73**	1.60**
2007	0.22***	0.38**	0.25***	0.76	0.32***	1.26	0.51*	5.85***	0.31**	0.58*	0.80	0.71**	2.01***

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$

CD: Communicable diseases; NCD: Non-communicable diseases; CoD: Cause of death; \$: Communicable diseases other than HIV/AIDS and Tuberculosis; £: Non-communicable diseases other than injury and cancer.

**All non-communicable diseases**

Overall men had a 45% higher risk of death from non-communicable diseases (includes deaths from injuries) than women. There were no significant differences by slum of residence. The Luhya, Cushites and Kamba ethnic groups were all less likely to die of disease in this category compared with Kikuyu ethnic group. Individuals in the second wealth tertile and the wealthiest were less likely to die of non-communicable diseases than the poorest individuals by over 30% while those of unknown wealth status were over 2.6 times more likely to die from non-communicable diseases. Primary school education and secondary or higher were associated with a 26% and 43% lower risk of death from all non-communicable diseases respectively.

**Non-HIV/AIDS causes of death**

Results show that men had a 30% higher risk of death from non-HIV/AIDS causes than women. Residents of Viwandani had a 28% lower risk of dying from non-HIV/AIDS compared with those of Korogocho. The Luhya and Kamba ethnic groups all had lower risk of dying from non-HIV/AIDS causes compared with Kikuyu while the Luo had 25% higher risk of dying from non-HIV/AIDS causes compared with the Kikuyu. The wealthiest and those in the middle tertile of the wealth ranking had over 30% lower risk of dying from non-HIV/AIDS causes than the poorest while those whose wealth ranking was not known had a 2.7 higher risk of dying from a non-HIV/AIDS cause. Secondary or higher education was associated with 40% lower risk of dying from a non-HIV/AIDS cause compared with no formal education. The risk of dying from a non-HIV/AIDS cause reduced in 2005 by 21%, in 2006 by 27% and in 2007 by 29%.

**Unknown causes**

Although this is not a valid cause of death category, I wanted to explore how the risk of death varied by the main socio-demographic characteristics used to examine the other known causes. This may give us some indication as to whether this category is unique and whether it requires further exploration to assess the extent of bias this might have on the estimates for the known causes of death. Men were 19% less likely to die of unknown cause than women. The Luo were about 50% more likely to die of an unknown cause than Kikuyu while the Kamba were 41% less likely. There was also significantly higher risk of dying from unknown causes in 2006 and 2007.

### **Exploration of potential interactions in the models in Table 16**

Table 17 gives hazard ratios after adjusting for interaction terms where any were found to be significant when put in the model one at a time. Models where no interaction terms were found to be significant retain results as contained in Table 16. In the model for HIV/AIDS, other communicable diseases, and cancer no interaction terms were found to be significant at 5% level of significance and therefore the results remain as those in Table 16 and no further discussion of the same is given here.

### **Tuberculosis**

The only significant interaction for tuberculosis was that of educational attainment and slum of residence. For residents of Korogocho slum, primary or higher education was associated with higher risk of death from tuberculosis than those with no education.

Among all those with no formal education, risk of death due to tuberculosis was about 2.5 times higher in Viwandani than Korogocho. However, the risk of death among residents of Viwandani from tuberculosis substantially decreased with education.

Viwandani residents with primary or secondary education had 17% and 66% lower risk than those of Korogocho with no formal education.

### **HIV/AIDS or Tuberculosis**

The significant interactions were sex and education; and slum and education. Risk of death among females with some education was higher than that among those with no formal education. It was highest among those with primary education. Among all those with no education, risk of death was higher among men by 13%. Compared with females with no formal education, males with some education had lower risk of death from either HIV/AIDS or tuberculosis with 60% lower risk among those with secondary or higher education. The same trend was observed for slum of residence and education. Risk of death among Korogocho residents was higher among those with some education while for Viwandani the risk was lower among those with some education as compared with residents of Korogocho with no formal education.

### **All communicable diseases**

Four interactions were significant including sex and slum; sex and education; slum and education; and slum and ethnicity. Residents of Viwandani with primary or secondary

education had significantly lower risk of death than those of Korogocho with no education. Men with primary or higher education had lower risk of dying from communicable diseases than female residents of Korogocho. Risk of death of Luo in Viwandani slum was about 4 times that of Kikuyu in Korogocho slum.

### **Injury**

Significant interactions were gender and slum and sex and calendar year. Male residents of Korogocho were about 6.5 times more likely to die of injury compared with female residents of Korogocho. On the other hand Viwandani men were about 4.4 times more likely to die of injuries than Korogocho women. Mortality from injuries among women for 2004, 2005 and 2007 was lower than that of 2003. Among men, for all the years, risk of death was at least 6.5 higher than of women in 2003 with highest risk at 9.4 times higher in 2007 and 9.5 times higher in 2004.

### **Non-Communicable diseases other than injury and cancer**

Significant interactions were sex and slum and gender and ethnicity. Male residents of Viwandani were about 49% less likely to die of diseases in this category than women of Korogocho. Among women those of Viwandani had about 38% higher risk of death from diseases in this category than Korogocho women. Risk of death from diseases in this category among men was lower than that of Kikuyu women.

### **All non-communicable diseases:**

Significant interactions were sex and wealth status; sex and education and sex and calendar year. Among females, the wealthiest had 11% lower risk of death from any of the non-communicable diseases than the poorest women while wealthiest men, had a 53% lower risk of death as compared with poorest women. Among women, risk of death decreased with educational level with a 43% and 69% lower risk among those with primary and secondary education respectively compared with women with no formal education. Among men, the risk of death from any of the non-communicable diseases decreased with increase in education but to a lesser extent as compared with the decrease among women. Differences in mortality from NCD between men and women were noticeable in 2003, 2004 and 2007.



Table 17: Hazard ratios from Cox proportional hazard regression models with interaction terms by cause of death

Variables	Hazard Ratios					Non-communicable diseases							
	Communicable diseases					Injuries							Non-HIV causes
	HIV/AIDS	Tuberculosis	HIV/TB	Other CD <sup>s</sup>	All CD	All injury	Un-intentional	Intentional injuries	Cancer	Other NCD <sup>£</sup>	All NCD	Missing CoD	
Gender													
Females	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Males	0.35***	0.94	1.13	1.18	1.47	6.54***	6.41***	9.26***	0.36***	0.93	0.86	1.45	1.24
Slum													
Korogocho	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Viwandani	1.26	2.53	1.48	0.57	0.94	1.8	0.82	0.63	1.18	1.38	0.79	0.95	1.11
Ethnicity													
Kikuyu	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Luhya	1.15	1.07	1.13	0.96	0.91	0.51**	0.48*	0.58	1.05	1.07	0.66**	0.78	1.19
Luo	2.60***	1.71*	3.00***	2.29**	2.50***	0.8	0.80	0.82	1.86	1.43	0.98	1.37	2.05***
Kamba	0.75	0.87	0.69*	0.56	0.58*	0.40***	0.48**	0.26***	1.29	0.78	0.55***	0.69	0.96
Cushites	0.14**	1.8	0.64	1.50	0.67	0.33*	0.20*	0.52	0.31	1.53	0.51**	1.19	0.67
Others	0.82	0.28	0.53*	0.30	0.68	0.49*	0.62	0.26*	1.09	0.16	0.54**	0.35*	1.19
Wealth													
Poorest	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Middle	1.17	0.74	1.04	0.78	1.02	0.62*	0.68	0.52	0.46	0.71	0.98	0.67**	1.08
Wealthiest	0.82	0.59*	0.76*	0.77	0.78*	0.47***	0.58*	0.33***	1.08	0.82	0.89	0.66***	1.17
Unknown	1.06	3.74**	1.75	1.80	1.79	2.76**	3.75***	1.21	1.49	1.94	3.56*	2.67***	1.34
Education													
No education	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Primary	0.99	1.64	1.48	1.41	1.49	0.77	1.41	0.31*	0.56	0.99	0.57**	0.82	1.05
Secondary & higher	0.75	1.51	1.29	0.70	1.28	0.57	1.03	0.24**	0.46	0.93	0.31***	0.59**	0.8
Unknown	1.33	0.99	1.67	1.28	1.38	1.17	2.49	0.23	1.94	0.28	0.55	0.98	0.73
Calendar year													
2003	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
2004	0.72*	1.19	0.81	1.20	0.86	0.3	1.47	0.17	0.60	0.95	0.71	0.94	0.96
2005	0.61***	0.74	0.63***	0.80	0.66***	0.43	0.75	1.83	0.59	0.79	0.76	0.82	1.45
2006	0.31***	0.63	0.37***	0.75	0.42***	1.11	0.49*	4.39**	0.51	0.59*	0.69	0.64*	1.52
2007	0.22***	0.39**	0.25***	0.76	0.32***	0.27	0.51*	5.85***	0.31**	0.57*	0.52*	0.48***	1.99**
Gender*slum													

Males#Viwandani		0.69*	0.37*		0.40**	0.66*	
Gender*ethnicity							
Male#Luhya	1.05				0.65	0.92	0.45*
Male#Luo	1.93*				0.69	0.86	0.58*
Male#Kamba	0.70				0.78	0.78	0.44**
Male#Cushites	4.56				0.21*	0.41**	0.72
Male#Others	0.54				4.73	1.47	0.33*
Gender*wealth							
Males#middle tertile						0.56*	
Males#third tertile						0.62*	
Males#unknown						0.63	
Gender*education							
Males#primary		0.46*	0.46**			1.76*	
Males#secondary		0.40*	0.37**			2.80**	
Males#unknown		0.47	0.53			2.51	
Gender*years							
Males#2004				4.86		1.74	1.24
Males#2005				2.31		1.10	0.94
Males#2006				0.92		1.16	1.23
Males#2007				5.33*		1.90*	1.81*
Slum*education							
Viwandani#primary	0.38*	0.20*	0.33**	0.49*			
Viwandani#secondary	0.44	0.09**	0.30**	0.44*			
Viwandani#unknown	0.43	0.67	0.47	0.79			
Slum*ethnicity							
Viwandani#Luhya				1.73			
Viwandani#Luo				1.72*			
Viwandani#Kamba				1.46			
Viwandani#Cushites				2.24			
Viwandani#Others				0.66			
Slum#years							
Viwandani#2004							0.67
Viwandani#2005							0.48*
Viwandani#2006							1.11
Viwandani#2007							1.02

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ . CD: Communicable diseases; NCD: Non-communicable diseases; CoD: Cause of death; \$: Communicable diseases other than HIV/AIDS and Tuberculosis; £: Non-communicable diseases other than injury and cancer.

### Non-HIV/AIDS

Three interactions were significant including sex and slum; gender and calendar year; and sex and ethnicity. Among females, resident of Viwandani had a 5% lower risk of death from non-HIV/AIDS related causes than those in Korogocho. Among all residents of Korogocho, Males had a higher risk (45%) of death from non-HIV/AIDS related causes than females. Viwandani males had 9% lower risk than Korogocho females. Kikuyu men had a 45% higher risk of death from non-HIV/AIDS than Kikuyu women while Luo men had 71% higher risk of death from non-HIV/AIDS related causes than Kikuyu women. Among women, mortality decreased over the years, but it only substantially decreased between 2004 and 2005 but remained higher than that for women in 2003.

### Multiple Imputations for missing cause of death data

Results in Table 18 show revised categories of causes of death used for imputation of missing cause of death. The table shows observed and imputed proportions contributed by the various causes of death. The observed data showed that HIV/AIDS accounted for about 34% of all cause of death while the imputed estimate was approximately 33%. In the observed data injuries contributed about 22% while in the imputed data injuries contributed 23%. Other communicable diseases other than HIV/AIDs and tuberculosis account for about 10% while the estimate in the imputed data was about 12%. Non-communicable cause of death other than injuries and cancer contributed about 18% in the observed data while in the estimate in the imputed data was 16%. In all causes of death, the observed and imputed proportions lie within confidence limits of each other.

Table 18: Percentage distribution of causes of death from observed and imputed data

Cause of death categories	Observed (N=1,071)		Imputed (N=1513)	
	Percentage	(95% CI)	Percentage	(95% CI)
HIV/AIDS	34.4	(31.5;37.2)	32.8	(29.5;36.1)
Tuberculosis	10.8	(9.0;12.7)	10.7	(9.0;12.3)
Other communicable	10.3	(8.4;12.1)	11.6	(9.7;13.4)
Injuries	21.6	(19.1;24.0)	23.4	(20.9;25.8)
Cancer	5.5	(4.1;6.9)	5.5	(4.1;6.8)
Other NCD	17.5	(15.2;19.7)	16.1	(13.5;18.7)

Table 19 (a) and Table 19(b) show the hazard ratios by cause of death with observed rates alongside the imputed rates for ease of comparison. The risk of death from HIV/AIDS by gender for the observed and imputed data was very similar (HR=0.45 from the observed data compared to 0.44 for the imputed data for males). By slum of residence imputed estimate (HR 0.56) was slightly higher than the observed (HR 0.52). The risk of death from HIV/AIDS among the Luo was about 2% lower in the imputed sample. The downward trend observed in the risk of death from HIV/AIDS between 2003 and 2007 in the observed and imputed data were comparable but generally lower in the observed data.

For tuberculosis, residents of Viwandani were about 49% less likely to die of tuberculosis as per observed data but the difference was insignificant in the imputed data. Similarly the risk of death due to tuberculosis in the observed data was about 62% lower in 2007 than 2003 but the difference was not significant in the imputed data. From the observed data, the Luo had a 73% higher risk of death, however the risk was lower (1.66) as per imputed data. In the categorisation of HIV/AIDS and tuberculosis, the estimates for gender, slum of residence and Luo and Kamba ethnic groups had significant comparable hazard rates in both observed and imputed data. The wealthiest individuals were significantly less likely to die of either HIV/AIDS or tuberculosis as per observed data but there was no significant difference in the imputed data. The significantly reducing trend in risk of death from either HIV/AIDS or tuberculosis in the observed data was mirrored in the imputed data though estimates were slightly higher in the imputed data.

Table 19 (a): Observed and imputed Hazard ratios from Cox proportional hazard regression models by cause of death

Variables	Cause-specific Hazard Ratios with levels of significance									
	HIV/AIDS		Tuberculosis		HIV/AIDS & Tuberculosis		Other CD <sup>s</sup>		All communicable diseases	
	Observed	Imputed	Observed	Imputed	Observed	Imputed	Observed	Imputed	Observed	Imputed
Gender										
Females	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Males	0.45***	0.44***	0.96	0.94	0.54***	0.53***	1.18	1.25	0.62***	0.49***
Slum										
Korogocho	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Viwandani	0.53**	0.57*	0.50*	0.52	0.53***	0.56**	0.57	0.74	0.53***	0.55***
Ethnicity										
Kikuyu	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Luhya	1.15	1.05	1.07	1.04	1.13	1.05	0.96	0.94	1.10	1.14
Luo	3.45***	3.32***	1.73*	1.66*	2.99***	2.89***	2.29**	1.88*	2.87***	3.00***
Kamba	0.62*	0.63*	0.83	0.92	0.68*	0.71*	0.56	0.45*	0.66**	0.72*
Cushites	0.30**	0.35**	1.63	1.31	0.63	0.61	1.50	1.34	0.82	0.69
Others	0.63	0.79	0.25	0.25	0.53*	0.64	0.30	0.18*	0.49**	0.51*
Wealth										
Poorest	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Middle	1.18	1.25	0.78	0.93	1.06	1.15	0.78	0.74	1.01	1.05
Wealthiest	0.83	0.91	0.60*	0.64	0.76*	0.84	0.77	0.85	0.76*	0.79*
Unknown	1.08	0.97	3.84**	3.44*	1.78	1.62	1.80	1.36	1.79	1.91*
Education										
No education	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Primary	0.84	0.91	1.08	1.09	0.90	0.96	1.41	1.25	0.99	0.92
Secondary or higher	0.67	0.74	0.72	0.77	0.69	0.75	0.70	0.68	0.70	0.69
Unknown	1.17	1.00	1.41	1.25	1.22	1.06	1.28	1.17	1.25	1.17
Calendar year										
2003	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
2004	0.72*	0.71*	1.19	1.18	0.81	0.80	1.20	1.22	0.86	0.83
2005	0.61***	0.71*	0.73	0.81	0.63***	0.73*	0.80	0.96	0.66***	0.66**
2006	0.31***	0.43***	0.62	0.85	0.37***	0.51***	0.75	1.00	0.42***	0.37***

2007	0.22***	0.41***	0.38**	0.68	0.25***	0.46***	0.76	1.33	0.32***	0.25***
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\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$   
*CD: Communicable diseases*  
*§: Communicable diseases other than HIV/AIDS and Tuberculosis*

Table 19 (b): Observed and imputed Hazard ratios from Cox proportional hazard regression models by cause of death

Variables	Cause-specific Hazard Ratios with levels of significance									
	Injury		Cancer		Other NCD <sup>‡</sup>		All NCD		Non-HIV/AIDS	
	Observed	Imputed	Observed	Imputed	Observed	Imputed	Observed	Imputed	Observed	Imputed
Gender										
Females	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Males	7.38***	6.09***	0.36***	0.32**	0.54***	0.51***	1.45***	1.35*	1.30**	1.25**
Slum										
Korogocho	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Viwandani	0.73	0.85	1.18	1.49	0.90	0.92	0.83	0.93	0.72**	0.82
Ethnicity										
Kikuyu	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Luhya	0.51**	0.44***	1.05	1.34	0.89	0.93	0.65**	0.65*	0.75*	0.74*
Luo	0.80	0.73	1.86	1.70	1.23	1.15	0.97	0.90	1.25*	1.15
Kamba	0.40***	0.37***	1.29	1.28	0.69	0.75	0.55***	0.54***	0.58***	0.58***
Cushites	0.33*	0.31*	0.31	0.24	0.90	0.88	0.49**	0.47**	0.74	0.69*
Others	0.48**	0.49*	1.09	1.34	0.51	0.49	0.53**	0.55**	0.47***	0.47***
Wealth										
Poorest	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Middle	0.63*	0.72	0.46	0.55	0.73	0.82	0.64**	0.73*	0.68**	0.76*
Wealthiest	0.47***	0.57**	1.08	1.28	0.82	0.94	0.66***	0.78	0.67***	0.77**
Unknown	2.76**	2.62**	1.49	1.78	1.87	1.55	2.56***	2.41**	2.66***	2.42***
Education										
No education	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Primary	0.79	0.93	0.56	0.61	1.03	1.06	0.71*	0.78	0.83	0.88
Secondary or higher	0.58	0.63	0.46	0.53	0.95	0.97	0.57**	0.60*	0.60**	0.63**

Unknown	1.19	1.19	1.94	1.63	0.28	0.23	0.87	0.81	0.97	0.92
Calendar year										
2003	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
2004	1.27	1.24	0.60	0.56	0.95	0.93	1.02	0.99	1.08	1.06
2005	0.90	0.94	0.59	0.66	0.79	0.79	0.80	0.83	0.79*	0.85
2006	1.04	1.43	0.51	0.67	0.59*	0.75	0.75	1.01	0.73**	0.98
2007	1.26	1.62*	0.31**	0.62	0.58*	0.84	0.80	1.12	0.71**	1.07

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$

*NCD: Non-communicable diseases; £: Non-communicable diseases other than injury and cancer.*

Risk of death from communicable diseases other than HIV/AIDS and tuberculosis among the Luo was attenuated from 2.29 in the observed data to 1.88 in the imputed data. Among the Kamba ethnic group the risk of death from communicable diseases other than HIV/AIDS and tuberculosis was not significantly different from that of the Kikuyu; however in the imputed data the risk was of comparable magnitude but significantly lower than that of the Kikuyu. For all communicable diseases, the imputed risk estimated for mortality by gender show that males had a 51% lower risk compared with females while in the observed data the risk was 38% lower among males. Estimates for ethnicity, wealth index, and education from both observed and imputed data were comparable. For both observed and imputed data, the risk of death by calendar year significantly decreased since 2005, although the reduction was more marked in imputed data.

Table 19(b) is a continuation of Table 19(a). Results in Table 19 (b) show that the risk of death from injury in the observed data was 7.4 times higher among males compared with females; however the imputed data show an attenuated risk of 6.1. While the observed data shows that the risk of death from injuries since 2003 has not significantly changed, the imputed data show that the risk of death from injuries in 2007 was significantly higher than in 2003. The observed data showed that the risk of death from cancer was significantly lower in 2007, while the imputed data that showed that the difference was not significant. Similarly while the risk of death from non-communicable diseases other than injuries and cancer in the observed data was significantly lower in 2006 and 2007 compared to 2003, the difference in the imputed data was not significant. Observed data showed that risk of death from all non-HIV/AIDS related causes was significantly lower in Viwandani compared with Korogocho and higher among the Luo compared with the Kikuyu. The imputed data estimates show that the differences were insignificant. From the observed data, significantly lower risk of death from all non-HIV/AIDS causes of death from 2005 to 2007 was observed but this was insignificant in the imputed data.



## 5.4 Discussion

Among deaths with cause-of-death data, HIV/AIDS was the leading cause of death with over 34% of all deaths being associated with it. Currently, the prevalence of HIV in Kenya is about 7.4% while in the two informal settlements it is 11.5% <sup>[13, 234]</sup>. Many sub-Saharan African countries are seeing a decline in the prevalence of HIV. In Kenya, however, prevalence has remained high and has not declined in the last 10 years <sup>[12, 13]</sup>. The true share of HIV/AIDS deaths in this population might be higher than was estimated here because, given the weaknesses of a verbal autopsy tool, some HIV/AIDS cases could have been classified under tuberculosis or other HIV/AIDS associated diseases such as pneumonia (pneumocystis carinii pneumonia) and meningitis (cryptococcal meningitis) <sup>[258, 259]</sup>. While this is possible, studies have shown that verbal autopsy tools have reasonably high sensitivity and specificity in identifying HIV/AIDS deaths especially among adults <sup>[72, 77, 260]</sup>. Also, from a public perspective, knowledge of broad disease entities probably represents adequate information for programming and, as such, the weakness of the verbal autopsy in identifying HIV/AIDS related deaths is not a compelling argument against use of the approach given the absence of a better alternative. Indeed, the scaling up of access to antiretroviral therapy is expected to reduce mortality due to HIV/AIDS; however in the absence of a system to monitor cause-specific mortality, the performance of such interventions will be difficult to measure.

Individuals that were identified as having died of tuberculosis accounted for about 11% of all causes of death. The HIV/AIDS epidemic has been associated with an upsurge of tuberculosis incidence and mortality <sup>[251, 261]</sup>. Tuberculosis is a common opportunistic infection in HIV-infected individuals and, as a result, the spread of HIV/AIDS has been accompanied by increase in tuberculosis infections <sup>[262]</sup>. Tuberculosis is a preventable and curable disease. However, the HIV/AIDS epidemic has had an impact on tuberculosis control efforts in terms of financial and human resources at a programmatic level. At the individual level, HIV/AIDS hastens progression from latent tuberculosis infection to active infection and hence produces an increase in tuberculosis cases and related deaths <sup>[251, 262]</sup>. Additionally, taking multiple HIV/AIDS and anti-tuberculosis drugs may impact adherence to regimens and thus lead to poor treatment outcomes.

HIV/AIDS and tuberculosis combined are a major public health challenge accounting for over 45% of all causes of death. An earlier study using the burden of disease approach in the same population estimated that, between 2003 and 2005, HIV/AIDS and tuberculosis accounted for over 50% of the years of life lost (YLL) due to premature death <sup>[1]</sup>. These findings are in line with several reports that have indicated that HIV/AIDS and tuberculosis are leading causes of death in many countries in Sub-Saharan Africa and thus need urgent attention <sup>[263]</sup>.

While injury as a cause of death is increasingly becoming a major public health challenge in sub-Saharan Africa, many authorities do not rank it among the top public health priorities <sup>[1, 264, 265]</sup>. The results of this study show that injury (both intentional and unintentional) was the second leading cause of death after HIV/AIDS. These results are similar to those reported in an earlier study of the same population that reported that, between 2003 and 2005, injuries accounted for about 18% of years of life lost due to premature death <sup>[1]</sup>. The disadvantaged nature of the slums in terms of housing, roads and other social amenities put slum residents at extra risk of injuries particularly from fires and other forms of unintentional injuries. The limited economic opportunities for slum residents have pushed many young men into alcohol and drug abuse, mainly of the illicit potent brews often laced with methanol. In addition, many youths have been mobbed or gunned down by the law enforcement agencies. Lastly, the traffic on the roads has increased with no corresponding increase in road capacity and this exposes motorists and pedestrians to increased risk of road traffic accidents <sup>[1, 266]</sup>.

Because nearly 30% of all deaths did not have a definitive cause of death, there is a possibility that those without cause of death data could be systematically different in important ways from those with cause of death data. And, if this were the case, it could potentially bias the contributions of specific causes. The missing causes of death partly arose as a result of ambiguity of signs and symptoms, which can also lead to misclassification of cause of death. For example, it has been reported that anticipated HIV/AIDS related deaths are associated with migration to rural destinations <sup>[105]</sup>. If this happened, the mortality estimate in the “sending” community may be underestimated. In this population out-migration is high and it is possible that some of this mobility is associated with ill-health. This point is made stronger by the observation that the Luo,

who have a higher risk of death from HIV/AIDS, also have a higher risk of not having cause-of-death data. However, the time period under consideration corresponds with the coming of life-improving and life-prolonging anti-retroviral treatment for HIV/AIDS. Antiretroviral therapy generally became more available in the urban areas earlier than the rural areas and, therefore, it would appear that the motivation for the terminally-ill individuals to migrate may not be as great as it was previously. The urban areas also offer more opportunities for involvement in the formal and informal labour market and thus continued support to the family. Assessment of potential bias using multiple imputation multivariate methods showed little difference between observed and imputed estimates. This implies that cases with no cause-of-death data were not systematically different from those with cause of death data with respect to the known risk factors measured in this study.

Although the verbal autopsy tool has proved useful in generating cause of death data where limited or no vital registration exists, it has limitations <sup>[267]</sup>. Its sensitivity and specificity for identifying certain diseases and in certain age groups has been reported to be low. For example, in a validation study, Kahn et al. found that the sensitivity and specificity for non-communicable diseases was 64% and 50% respectively while that for communicable diseases was 89% and 93% <sup>[76]</sup>. The observation here, that individuals above 75 years of age were less likely to have a cause of death could partly be attributed to the tendency of many causes of death in old age, such as cardiovascular conditions, dementia and multiple organ failure, to be difficult to diagnose using verbal autopsy as they tend to present with non-specific symptoms. The potential effect of this in this population is underestimation of non-communicable diseases, which are more common in old age.

Regarding wealth, it is not clear why the wealthiest individuals were less likely to have cause-of-death data. It is important to note is that, since 2005, more and more deaths have not had a cause of death assigned to them. It is hard to find an explanation for this, but it could be related to respondent fatigue, with continued participation in the health and demographic surveillance leading to higher levels of refusals. It is unclear whether this trend could be associated more with certain causes of death than others. Because individuals classified as highly mobile might have a distinctive risk profile of disease,

for example HIV among truck drivers, I attempted to control for mobility. Highly mobile individuals did not have significantly different chances of missing cause of death data than those categorised as less mobile. While this allays some fears, the mobility index may not capture mobility associated with risk of disease, such as short periods of absence from home, as opposed to the changes of residence within or without the slum used in this analysis.

After adjusting for age, HIV/AIDS remained the leading cause of death overall and was followed by injuries. Sex-specific death rates showed that HIV/AIDS was the leading cause of death among females while injuries were the leading cause among males. Among women, it was surprising to see cancer and diabetes mellitus in second and third position as causes of death –coming before maternal causes. However, it is important to note that fertility in this population is relatively low and therefore the contribution of maternal health conditions is expected to be low. Also surprising among women was the ranking of injury among the top 10 causes of death, indeed coming before maternal causes, although the published literature has shown that the maternal mortality ratio in this population is as high as 706 maternal deaths per 100,000 live births of which 30% are attributable to abortion complications <sup>[96]</sup>. Among men, injuries were the leading cause of death followed by HIV/AIDS, tuberculosis and cardiovascular diseases in that order. These results show that while communicable diseases, namely HIV/AIDS and tuberculosis, are taking a huge toll on this population, there is an emerging challenge of non-communicable diseases particularly injuries, diabetes mellitus and cardiovascular diseases. Currently most public health resources are being directed, and rightly so, towards HIV/AIDS and tuberculosis programmes, but, as the results show, the emerging challenge of non-communicable diseases should be addressed early.

Modelling risk of death by cause of death revealed that risk of death due to HIV/AIDS among men was lower than that among women. This finding is in line with expectation because both national and slums estimates of HIV show that prevalence of HIV is higher among females <sup>[12, 13, 234]</sup>. Generally, women tend to have earlier access to care, better adherence to treatment, and slower progression to AIDS than men. The resultant better survival for HIV infected women might partly explain the higher HIV prevalence among women in the period under study <sup>[268, 269, 270, 271, 272]</sup>.

The risk of death due to HIV/AIDS among residents of Viwandani was lower than that of Korogocho by nearly 50%. It is not clear why these two communities have markedly different risk profiles of HIV prevalence and death due to HIV/AIDS. Mortality results are consistent with the results of an earlier study showing that HIV prevalence in Viwandani was 8% compared to 14% in Korogocho <sup>[234]</sup>. Similarly, the Luo ethnic group had more than 3 times the risk of death compared with the Kikuyu, while other ethnic groups had similar or lower risk of death than the Kikuyu. This reflects the high HIV prevalence rate among the Luo <sup>[12, 13]</sup>. Wealth and education status did not have any influence on mortality due to HIV/AIDS. The downward trend in mortality observed over the years is more marked for deaths due to HIV/AIDS and starts as early as 2003. While tuberculosis and HIV co-infections are common and are likely have similar patterns, results showed that there were no gender differences in risk of death due to tuberculosis, suggesting that the verbal autopsy tool may have been able to distinguish HIV/AIDS from tuberculosis as cause of death. The time trend in the risk of death due to tuberculosis was not as marked as that of HIV/AIDS and was only significant in 2007. The causes of death grouped together as communicable diseases other than HIV/AIDS and tuberculosis had a similar pattern as HIV/AIDS – a lower risk of death among men, and residents of Viwandani and a downward trend over time. This might be related to the fact that some of the diseases in this category occur together with HIV/AIDS as opportunistic infections particularly cryptococcal meningitis and pneumocystis carinii pneumonia. If this was the case, it means that the mortality burden attributable to HIV was underestimated.

The risk of death from injuries, all non-communicable diseases, and all non-HIV/AIDS related causes was higher among men than women. This means that the reversal of mortality –higher mortality among women- is mainly attributable to communicable diseases. Although the category of cancer is not broken down by the specific types of cancer, there are indications that cervical cancer plays a big role in the female cancer mortality and this in turn is associated with HIV infection, especially among young women <sup>[273]</sup>. The high risk of death among the Luo ethnic group is also limited to communicable diseases. It is not clear why the Luo have excess mortality due to other communicable diseases but it could be related to HIV/AIDS. The marked downward

trend in the risk of death seen with HIV/AIDS is not reflected in mortality due to injuries and is also not as marked for the non-communicable diseases.

### **Conclusions**

HIV/AIDS, injuries and tuberculosis are the leading causes of adult death. There also appears to be a downward trend in mortality primarily driven by a reduction in HIV/AIDS related mortality, particularly, among women. The Luo ethnic group has the highest risk of death particularly from HIV/AIDS, but the Kikuyu have a higher risk of death from injuries. While communicable diseases are the predominant causes of deaths, injuries and other non-communicable diseases are also important causes of death and these need to be given due consideration early before they get out of hand.

## **6. Impact of adult death on child migration**

### **6.1 Introduction**

This chapter concerns the migration of children aged 0-14 years living in two informal settlements of Korogocho and Viwandani with a specific focus on the effect of adult death. Migration in the African context has mainly been studied in relation to work-related movements of adults. As a result, data on the profiles, motivations for and consequences of migration among men and women are widely available <sup>[99, 111, 274, 275]</sup>. Less attention has been paid to the migration of children. One of the underlying assumptions of much of the literature on migration is that children tend to be non-migrants or if they migrate, they tend to do so with their parents, as a family unit <sup>[276]</sup>. Most studies of child migration have highlighted the plight of abused and neglected children, trafficked children, children left behind by migrant parents and - to a lesser extent - orphaned children <sup>[276, 277, 278]</sup>. The available evidence, however, shows that children are increasingly involved in migration streams, and in many cases, without their parents. Examples of situations driving child migration may include; orphanhood, family breakdown, and lack of support, economic hardships, school placement and forced migration due to insecurity as is often the case in areas under civil conflict <sup>[102, 278, 279]</sup>.

The population of the informal settlements is mainly composed of rural-urban migrants who predominantly come into the city in search of employment <sup>[100]</sup>. Not only do slum residents frequently change place of abode within the same slum, they also move between slums, other towns and rural areas. On the other hand, while there is a lot of movement within and between slums and back and forth between urban and rural areas, many residents live in the city long term and a sizeable proportion of children living in slums were born there <sup>[100]</sup>. Migration research in the two slums has generally shown that long-term residents have lower chances of subsequent out-migration than recent migrants. Of the two slums studied, Korogocho has a lower incidence of migration than Viwandani, and women have higher migration rates than men <sup>[100]</sup>.

Understanding child migration and its drivers is important as migration may impact on children's health and other social outcomes and development <sup>[122, 280]</sup>. The consequences

of, and vulnerability brought about by, prime-age death such as loss of care, support and nutrition may act as a push factor leading affected children to migrate as a response to the hardships encountered <sup>[19, 281]</sup>. While the usage of the term prime-age in other studies refers to various age categories, for purposes of this thesis in the methods, results and discussion prime-age is used to refer to adults 15-59 years of age.

While the body of literature relating child migration to adult mortality, mainly in the context of HIV/AIDS, is growing, no research has been carried out that examines children's migration patterns and the impact of adult death in a setting similar to the slums in Kenya <sup>[24, 109]</sup>. These movements are potentially disruptive and detrimental to children's health, education and socialisation as has been shown in studies elsewhere <sup>[111, 282]</sup>. Much as migration might be seen as an adjustment strategy, it might not necessarily be beneficial to children. In the context of high prime-age mortality and an ever-growing number of orphaned and vulnerable children, and a less socially cohesive community, children who lose guardians do not have many options of appropriate households to go to and thus their migration might lead them to worse circumstances <sup>[1, 4, 33, 111]</sup>.

The extended family structure, which is common in rural areas, is quite limited in the urban areas. As such, children who leave their families due to death of their guardians are likely to live in unfavourable circumstances such as living with non-economically active elderly people or with under-age siblings. In the extreme, affected households may dissolve altogether, with children ending up as street children or living with non-relatives.

This chapter seeks to examine the migration patterns of children living in Korogocho and Viwandani and assess how death of an adult affects the likelihood of child migration. Two forms of migration are examined: intra-slum change of residence and out-migration to destinations outside of the slum. It is hoped that the results from this chapter will help inform policy with respect to orphaned and other vulnerable children in marginalised communities. The main hypothesis being tested is: children who lived in households that experienced an adult death were more likely to migrate than those who lived in households that did not experience any adult death.



## 6.2 Methods and data sources

Analysis was restricted to children aged 0-14 years. Descriptive statistics of children's migration experience in the two slums are given for those children who lived in households that were exposed and those who lived in household that were not exposed to adult death. Descriptive statistics are also presented by slum and calendar year.

Aspects of migration examined include:

- i) Out-migration: This refers to a registered resident moving out of the demographic surveillance area with or without other household members for a period of 120 days or more;
- ii) Internal migration: This refers to change of residence of a registered resident within the slum. It involves a member leaving the original household within the slums to join or form a new household within the slum and living there for at least 120 days;

In the multivariate analyses, the main explanatory variable was exposure to adult death categorised as no adult death, father died, mother died or other adult died. The two main outcome variables, out-migration and internal change of residence, are dichotomous. Control variables included household socio-economic status measured in tertiles using household assets, household size categorised as  $\leq 3$ ; 4-5 and 6 or more members, gender of household head and child's relation to household head, child's sex, age, ethnicity, maternal education and slum of residence. Socio-economic status, household size and child's relation to household head are all time-varying variables.

The events for each of the two outcomes considered in the analyses can occur more than once for a given child. Accordingly, both fixed and random effects models were fitted to assess the relationship between adult death and the key outcome variables controlling for socio-demographic and household characteristics. Results from the two models were compared using Hausman test which tests the hypothesis that the coefficients estimated by the more efficient random effects model are the same as the ones estimated by the consistent but less efficient fixed effects model. As a general recommendation, a significant result indicates that one should use a fixed effects model. In these analyses, Hausman tests were significant on all outcomes. However, results from both fixed effects and random effects models are presented to allow for an assessment of the two

models beyond what the Hausman test tells us. Significant differences in the coefficients from the two models may be due to omitted unobserved child variables in the random effects specification or an incorrect assumption that the error term is not correlated with the predictors. Fixed effects models on the other hand control for unmeasured within-child time-invariant variables but make the assumption, which may not be valid, that unmeasured variables are time invariant. Additionally, estimates from the fixed effects model cannot be extrapolated to a wider population beyond the sample on which it is fitted. The random effects model has the extra appeal in that, in addition to giving estimates for time-varying covariates, it also gives estimates for time-invariant covariates which might be important predictors of the outcome <sup>[283]</sup>.

### **6.3 Results**

High mobility is a key characteristic of residents in the two slums examined. Figure 10 shows the proportion of children aged 0-14 years who remained residents of the slums over time. Proportions were computed as a fraction of children present in a preceding year that remained resident in the current calendar year. This was done for each “calendar year population cohort”. Results show that the proportions of children who migrated out of the various calendar year population cohorts were similar. For the 2003 cohort, at the end of the five year period (2007), about 50% of the original cohort of children had out-migrated out of the slums. The highest proportion lost in a single calendar year was about 25% in 2005.

Figure 10: Population cohort attrition of children who were aged 0-14 years in the Korogocho and Viwandani slums.

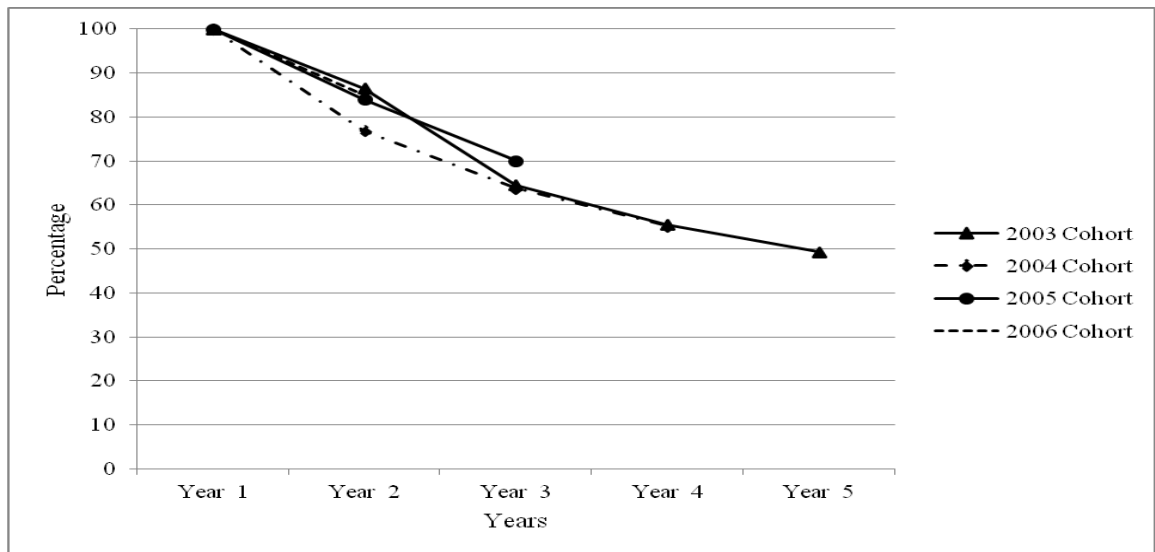


Figure 11 shows proportions of children who migrated away from the surveillance area by exposure status and calendar year. In 2003, of all children aged 0-14 years who lived in the slums, about 13.5% out-migrated in the same year. The corresponding percentages for the two slums were 11.4% and 16% for Korogocho and Viwandani respectively. The proportions of those who out-migrated from households that had been exposed to an adult death were slightly lower than those from households with no adult death. In the calendar year 2004, the proportion of children who out-migrated was higher (32%) than the other years. For the year 2005 and 2007, the proportion of children who out-migrated was about 16% while that of 2006 was about 15% with minor differences by exposure status and slum of residence.

Figure 12 shows proportions of children who had internal change of residence (within the slums) by exposure status and calendar year. In all the years, the proportion of children who changed place of residence within the slums varied in a narrow range of 14.5% in 2003 and 17.7% in 2006. The proportions were generally higher among those who lived in households that had experienced an adult death. This difference was more pronounced in Viwandani than in Korogocho slum. The highest proportion of children who changed place of residence was in exposed households in 2004, 2006 and 2007, all in Viwandani slum.

Figure 11: Proportion of children 0-14 years who out-migrated from surveillance area by exposure status and calendar year 2003-2007.

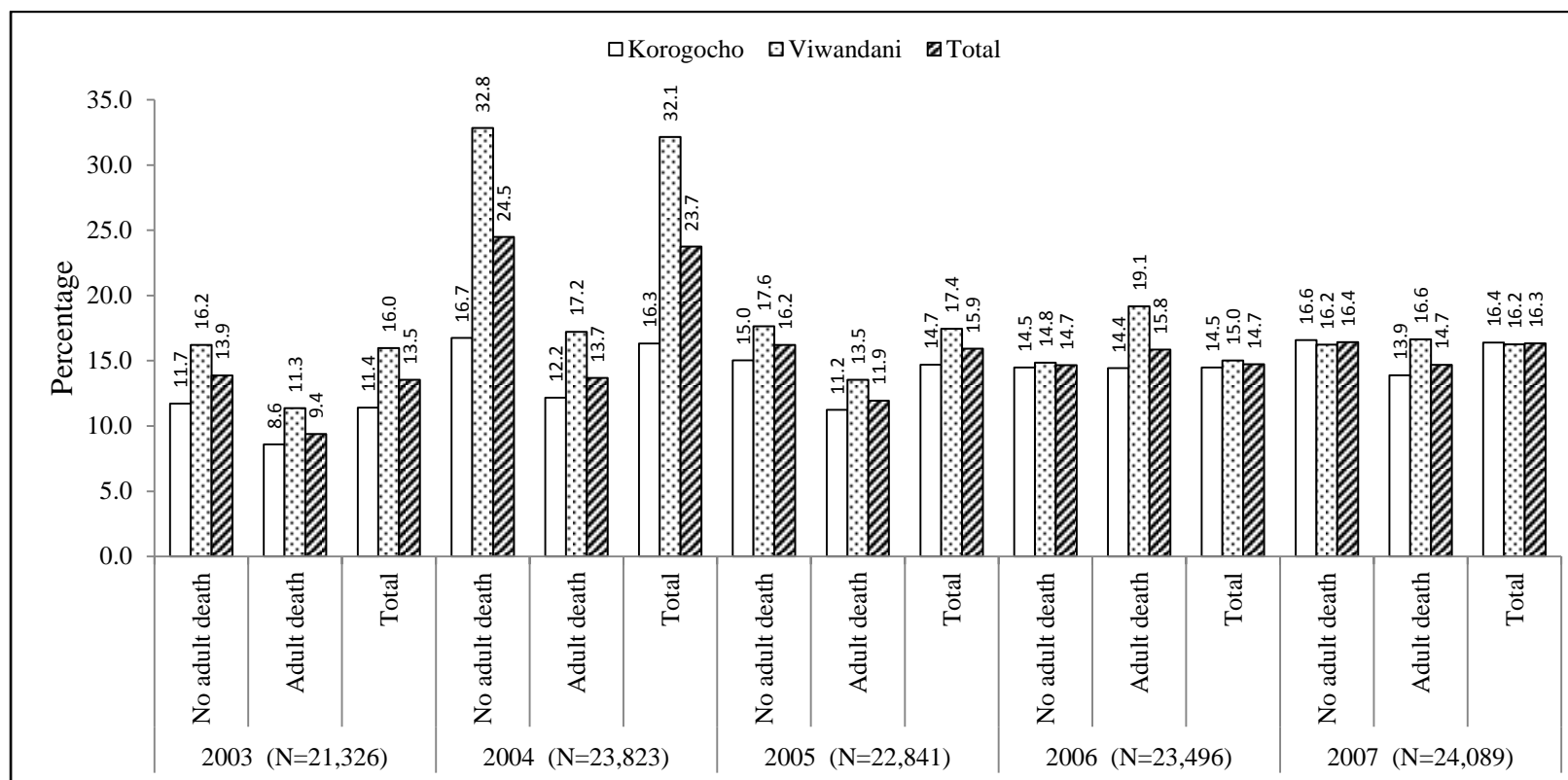
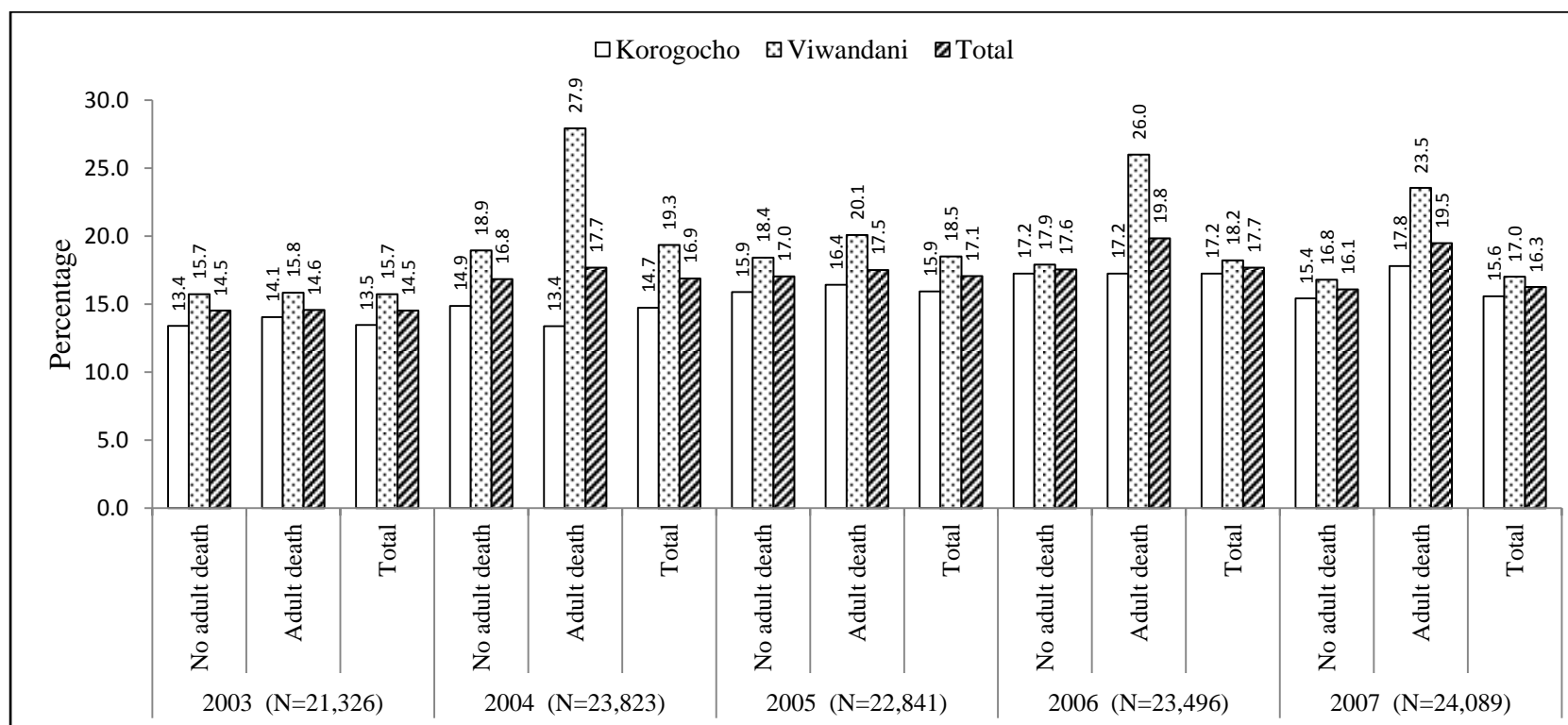


Figure 12: Proportion of children 0-14 years who had internal change of residence by exposure status and calendar year



### **Multivariable analysis for out-migration**

Table 20 shows results from random and fixed effects models examining the relationship between death of an adult and child out-migration controlling for household and individual child socio-demographic variables. The table has both unadjusted and adjusted estimates. The models with an interaction between exposure and household size provided a better fit based on the likelihood ratio test and therefore results from the models with interaction terms are also presented.

From the random effects model, the odds of child out-migration were lower for children who lost a non-mother adult in a household compared with those who lived in households that did not experience an adult death. Conversely, the odds of out-migration for children who lost a mother were significantly higher (OR=1.23; p value <0.05) than for children who lived in households with no adult death. Estimates from the model with interaction term between exposure and household size show that, in the small households, death of a non-mother adult was associated with higher odds of out-migration although this was insignificant while for death of a mother, the odds of child outmigration were higher (OR=2; p value<0.001). For medium sized households, death of a non-mother adult was associated with generally lower odds of child migration while for that of a mother the odds were close to unity compared with unexposed children living in small households (derived by multiplying out the estimates from the interaction model). For large households, death of any adult was associated with lower odds of child out-migration compared with children from unexposed households.

In the fixed effects models specification, the estimates are generally higher than those observed in the random effects models. Estimates indicate that death of a father was associated with lower odds of out-migration while death of a mother was associated with higher odds of out-migration although these were not statistically significant. Death of non-parent adults was associated with 2.5 times higher odds of out-migration. In the model with interactions, it can be seen that in small households, death of any adult was associated with higher odds of out-migration than those observed in the random effects model. In medium sized households, death of a father was associated with 48% lower odds of out-migration while that of a mother was associated with only

3% higher odds of out-migration. In large households, death of any adult was associated with lower odds of out-migration compared with children from small and unexposed households.

Other predictors of out-migration are noted here. In the random effects model, holding other factors constant, children living in households where the household head was a non-prime age individual had about 30% lower odds of out-migration compared with children living in households headed by a prime-age individual. However in the fixed effects model, the association between exposure and outcome was in the opposite direction. Children from households with a non-prime age head had about 50% higher odds of out-migration. Furthermore, results from the random effects models show that children living in households where the household-head was a non-parent or male had higher odds of out-migration than those who lived in households where the head was a parent or a female. In the fixed effects models, the estimates for non-parent head were close to 1 and insignificant while those for sex of household head were significant but in the reverse direction to those estimated by the random effects model.

In the random effects model, the odds of child out-migration decreased with increase in child's age, with 9% lower odds for those aged 4-9 years and 31% lower odds for those aged 10 to 14 years as compared with children aged less than 5 years. The results in the fixed effects models were in the opposite direction with increasing odds of out-migration with increase in child age. Children living in Viwandani slum had approximately 39% higher odds of out-migration than their Korogocho slum counterparts. The odds of child out-migration among all identifiable ethnic groups were higher than that of the Kikuyu ethnic group. In the random effects model, children from middle level income households had lower odds of out-migration than the poorest while estimates from the richest households in the random effects model show that they had marginally higher odds of out-migration than the poorest.

Table 20: Factors associated with children's out-migration from the slums: results from random and fixed effects logistic regression models

Variables	Random effects models			Fixed effects models		
	Unadjusted OR (95% CI)	Adjusted OR (95% CI)	With exposure * HH size interaction	Unadjusted OR (95% CI)	Adjusted OR (95% CI)	With exposure * HH size interaction
Exposure						
Not exposed	1.00	1.00	1.00	1.00	1.00	1.00
Father died	0.53*** (0.42,0.66)	0.63*** (0.51,0.79)	1.21 (0.78,1.90)	0.73 (0.41,1.28)	0.75 (0.42,1.34)	3.10* (1.00,9.60)
Mother died	1.02 (0.85,1.21)	1.23* (1.03,1.45)	2.00*** (1.45,2.75)	1.95* (1.02,3.72)	1.42 (0.72,2.80)	5.09** (1.94,13.38)
Other adult died	0.60*** (0.53,0.68)	0.74*** (0.66,0.84)	1.23 (0.98,1.56)	3.17*** (2.11,4.78)	2.52*** (1.66,3.82)	18.61*** (7.20,48.07)
Household head age						
Prime-age	1.00	1.00	1.00	1.00	1.00	1.00
Child/elderly (<15/ 59+ )	0.56*** (0.52,0.59)	0.70*** (0.65,0.75)	0.70*** (0.66,0.75)	1.71*** (1.38,2.12)	1.53*** (1.22,1.93)	1.55*** (1.23,1.96)
Child's relation to HHH						
Parent	1.00	1.00	1.00	1.00	1.00	1.00
Non-parent	1.08** (1.03,1.14)	1.42*** (1.34,1.50)	1.42*** (1.34,1.50)	1.16 (0.95,1.40)	1.05 (0.84,1.30)	1.05 (0.84,1.31)
Sex of HHH						
Female	1.00	1.00	1.00	1.00	1.00	1.00
Male	1.23*** (1.18,1.28)	1.19*** (1.14,1.24)	1.19*** (1.14,1.24)	0.60*** (0.52,0.70)	0.73*** (0.62,0.85)	0.75*** (0.64,0.88)
Household size						
<=3	1.00	1.00	1.00	1.00	1.00	1.00
4-5	0.78*** (0.76,0.82)	0.77*** (0.74,0.80)	0.78*** (0.75,0.81)	0.87** (0.79,0.96)	0.87** (0.79,0.96)	0.92 (0.83,1.02)
6 and above	0.46*** (0.44,0.48)	0.52*** (0.50,0.55)	0.54*** (0.52,0.57)	0.76*** (0.67,0.86)	0.74*** (0.65,0.84)	0.82** (0.72,0.94)
Age category for child						
0-4 yrs	1.00	1.00	1.00	1.00	1.00	1.00
5-9 yrs	0.79*** (0.76,0.81)	0.91*** (0.87,0.94)	0.90*** (0.87,0.94)	3.15*** (2.82,3.52)	3.15*** (2.82,3.52)	3.15*** (2.82,3.52)
10-14 yrs	0.57*** (0.54,0.59)	0.69*** (0.66,0.73)	0.69*** (0.66,0.73)	13.48*** (11.01,16.51)	13.24*** (10.80,16.24)	13.17*** (10.74,16.16)
Sex of child						
Female	1.00	1.00	1.00			
Male	0.99 (0.96,1.03)	0.98 (0.95,1.01)	0.98 (0.95,1.01)			
Slum						
Korogocho	1.00	1.00	1.00			
Viwandani	1.42*** (1.37,1.46)	1.39*** (1.32,1.46)	1.39*** (1.32,1.46)			
Ethnicity						
Kikuyu	1.00	1.00	1.00			

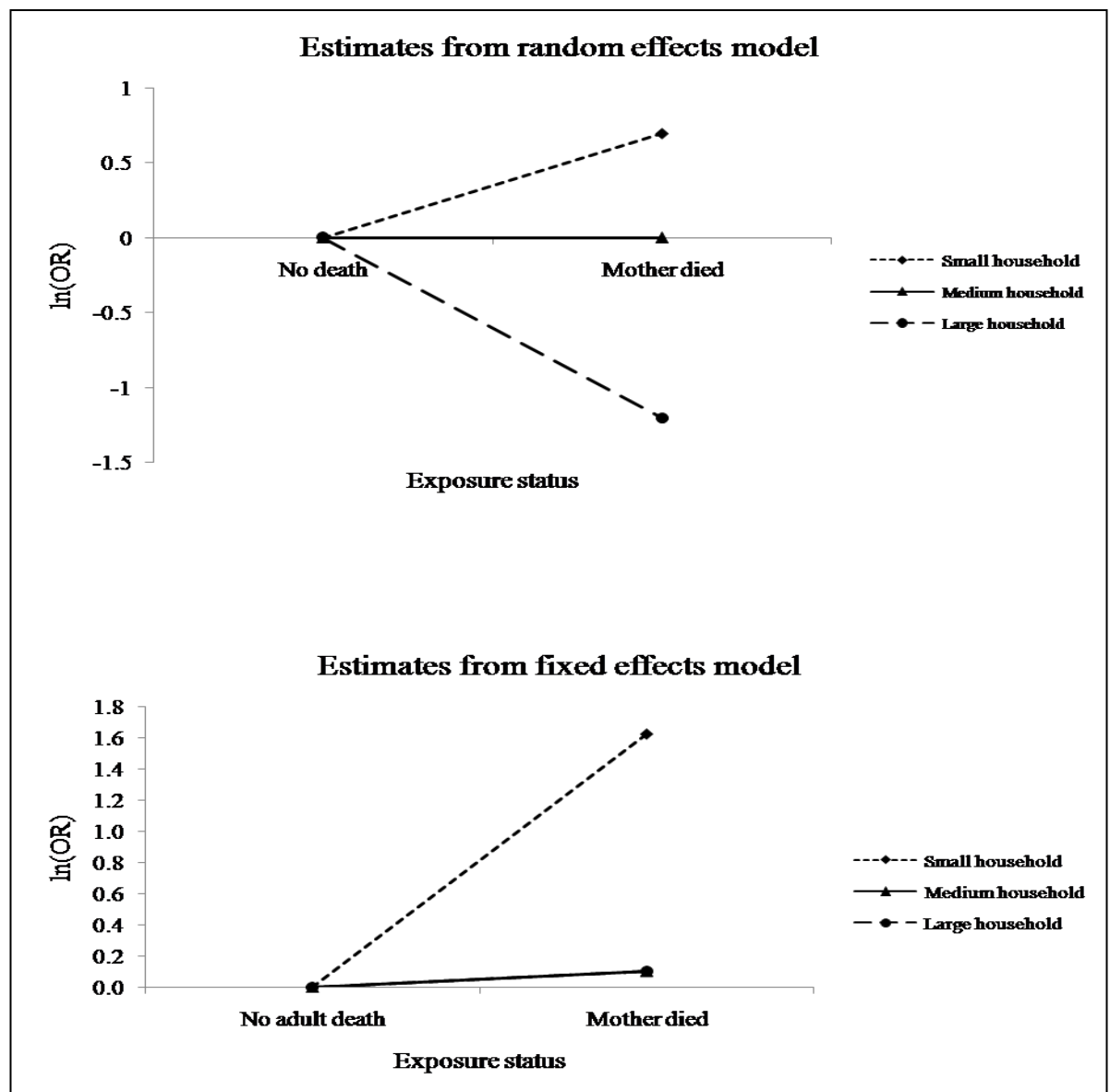


Luhya	1.62*** (1.54,1.70)	1.58*** (1.50,1.66)	1.58*** (1.50,1.66)			
Luo	1.56*** (1.49,1.64)	1.67*** (1.59,1.75)	1.66*** (1.58,1.74)			
Kamba	1.86*** (1.77,1.95)	1.55*** (1.47,1.63)	1.55*** (1.47,1.63)			
Other	1.00 (0.95,1.06)	1.17*** (1.11,1.24)	1.17*** (1.11,1.24)			
Wealth						
Poorest	1.00	1.00	1.00	1.00	1.00	1.00
Middle	0.97 (0.93,1.01)	0.80*** (0.76,0.83)	0.80*** (0.76,0.83)	0.67 (0.30,1.49)	0.68 (0.29,1.59)	0.66 (0.28,1.54)
Wealthiest	0.84*** (0.81,0.88)	1.04 (0.99,1.09)	1.04 (0.99,1.09)	0.73 (0.45,1.19)	0.60* (0.36,0.99)	0.61 (0.37,1.01)
Maternal education						
None	1.00	1.00	1.00	1.00	1.00	1.00
Primary	2.70*** (2.48,2.93)	1.76*** (1.61,1.92)	1.77*** (1.62,1.93)	2.18** (1.21,3.92)	2.21** (1.22,4.00)	2.33** (1.28,4.24)
Secondary/higher	2.68*** (2.45,2.92)	1.68*** (1.53,1.84)	1.68*** (1.53,1.84)	2.45** (1.25,4.78)	2.51** (1.26,5.00)	2.60** (1.30,5.19)
Unknown	3.12*** (2.83,3.44)	1.99*** (1.80,2.21)	1.99*** (1.80,2.21)	2.73** (1.47,5.09)	2.08* (1.08,4.00)	2.21* (1.14,4.26)
<b>Exposure X household size</b>						
Father died/medium HH size			0.55* (0.32,0.95)			0.31 (0.09,1.05)
Father died/large HH size			0.29*** (0.15,0.55)			0.06** (0.01,0.32)
Mother died/medium HH size			0.66* (0.44,0.97)			0.25** (0.11,0.55)
Mother died/large HH size			0.27*** (0.15,0.47)			0.09*** (0.03,0.29)
Other adult died/medium HH size			0.59** (0.43,0.81)			0.13*** (0.05,0.34)
Other adult died/large HH size			0.45*** (0.33,0.62)			0.06*** (0.02,0.16)
<i>Constant</i>		0.10*** (0.09,0.11)	0.10 (0.09,0.12)			
<i>Number of observations</i>		115575	115575		38585	38585
<i>Number of groups</i>		41305	41305		13693	13693
<i>Rho</i>		8.76E-06	8.78E-06			

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$

Figure 13 illustrates the interaction effects between exposure and household size only highlighting estimates for death of a mother. The figure shows that in large households, death of a mother lowers the risk of child out-migration. On the contrary, death of a mother in small sized households led to significantly higher odds of out-migration than those who lived in non-exposed households of the same size.

Figure 13: Risk of child out-migration: Interaction between death of a mother and household size.



### **Multivariable analysis of intra-slum migration**

Table 21 shows results from both random and fixed effects models examining the relationship between death of an adult in a household and the likelihood of a child changing place of residence within the slums. Unadjusted estimates are presented alongside adjusted estimates for the main exposure and other covariates for both random and fixed effects models. Furthermore, models with interaction term of exposure and household size are also presented.

Results from the random and fixed effects models showed that children exposed to an adult death were less likely to change place of residence than children living in households that did not experience an adult death. The estimates in the fixed effects model were attenuated compared with those from the random effects model. Estimates from the interaction terms (calculation not shown) in the random and fixed effects models show that in small households the likelihood of a child who lost a mother changing residence was 16% higher than that of a child living a small household and did not lose any adult in the household. In similar households, the risk of change of residence for those who lost a father or other adult was 45% lower and 18% higher respectively. In both medium and large sized households the likelihood of changing residence was lower among children who had been exposed as compared with those not exposed. Generally, the likelihood of changing residence decreased with increase in household size in both random and fixed effects models.

Children living in households headed by non-prime age individuals were less likely than others to change residence though this was not significant in the fixed effects model. There were no significant differences in the odds of a child changing place of residence within the slums for children living in households where the household head was a parent or non-parent. In the random effects model but not the fixed effects model children living in a male-headed household were less likely to change residence than those in a female-headed household. In both models, the likelihood of changing residence decreased with child's age.

Table 21: Factors associated with children's change of residence within the slums: results from random and fixed effects logistic regression models

Variables	Random effects models			Fixed effects models		
	Unadjusted OR (95% CI)	Adjusted OR (95% CI)	With exposure * HH size interaction	Unadjusted OR (95% CI)	Adjusted OR (95% CI)	With exposure * HH size interaction
Exposure						
Not exposed	1.00	1.00	1.00	1.00	1.00	1.00
Father died	0.32*** (0.23,0.46)	0.40*** (0.28,0.56)	0.55 (0.23,1.28)	0.13*** (0.08,0.21)	0.13*** (0.08,0.22)	0.21* (0.06,0.70)
Mother died	0.55*** (0.42,0.73)	0.63*** (0.48,0.82)	1.16 (0.71,1.90)	0.26*** (0.15,0.45)	0.28*** (0.16,0.48)	0.93 (0.38,2.28)
Other adult died	0.67*** (0.57,0.78)	0.68*** (0.58,0.80)	1.18 (0.86,1.63)	0.37*** (0.28,0.51)	0.44*** (0.32,0.60)	0.51* (0.30,0.87)
Household head age						
Prime age	1.00	1.00	1.00	1.00	1.00	1.00
Child/elderly (<15/ 59+ )	0.83*** (0.77,0.89)	0.85*** (0.78,0.92)	0.85*** (0.78,0.92)	0.82* (0.70,0.96)	0.91 (0.77,1.08)	0.91 (0.77,1.08)
Child's relation to HHH						
Parent	1.00	1.00	1.00	1.00	1.00	1.00
Non-parent	0.93* (0.87,0.99)	0.96 (0.89,1.03)	0.96 (0.89,1.03)	0.89 (0.78,1.02)	0.91 (0.78,1.06)	0.92 (0.79,1.07)
Sex of HHH						
Female	1.00	1.00	1.00	1.00	1.00	1.00
Male	0.81*** (0.77,0.85)	0.76*** (0.72,0.81)	0.76*** (0.72,0.81)	1.11 (0.99,1.24)	1.03 (0.91,1.16)	1.03 (0.91,1.16)
Household size						
<=3	1.00	1.00	1.00	1.00	1.00	1.00
4-5	1.00 (0.95,1.06)	1.07* (1.02,1.14)	1.09** (1.03,1.15)	1.12* (1.02,1.23)	1.15** (1.04,1.27)	1.17** (1.06, 1.29)
6 and above	0.85*** (0.80,0.90)	0.97 (0.91,1.03)	1.00 (0.93,1.06)	1.08(0.96,1.21)	1.17* (1.03,1.32)	1.21** (1.06,1.37)
Age category for child						
0-4 yrs	1.00	1.00	1.00	1.00	1.00	1.00
5-9 yrs	0.95* (0.91,1.00)	0.96 (0.91,1.01)	0.95 (0.91,1.00)	0.53*** (0.48,0.58)	0.54*** (0.49,0.59)	0.53*** (0.49,0.59)
10-14 yrs	0.82*** (0.77,0.86)	0.83*** (0.78,0.88)	0.83*** (0.78,0.88)	0.30*** (0.26,0.35)	0.31*** (0.27,0.36)	0.31*** (0.27,0.36)
Sex of child						
Female	1.00	1.00	1.00			
Male	1.01 (0.97,1.05)	1.01 (0.97,1.06)	1.01 (0.97,1.06)			
Slum						
Korogocho	1.00	1.00	1.00			
Viwandani	1.22*** (1.17,1.27)	1.08* (1.01,1.16)	1.08* (1.01,1.16)			

Ethnicity						
Kikuyu	1.00	1.00	1.00			
Luhya	0.99 (0.92,1.05)	1.07* (1.00,1.14)	1.07* (1.00,1.14)			
Luo	0.84*** (0.79,0.89)	0.93* (0.87,0.99)	0.93* (0.87,0.99)			
Kamba	0.98 (0.92,1.05)	0.94 (0.88,1.00)	0.94 (0.88,1.00)			
Other	1.20*** (1.13,1.28)	1.22*** (1.14,1.31)	1.22*** (1.14,1.31)			
Wealth						
Poorest	1.00	1.00	1.00	1.00	1.00	1.00
Middle	0.98 (0.93,1.04)	0.96 (0.90,1.02)	0.96 (0.90,1.02)	0.62 (0.37,1.05)	0.64 (0.37,1.09)	0.63 (0.37,1.09)
Wealthiest	0.79*** (0.75,0.83)	0.84*** (0.79,0.89)	0.84*** (0.79,0.89)	0.31*** (0.21,0.45)	0.34*** (0.23,0.51)	0.35*** (0.23,0.52)
Maternal education						
None	1.00	1.00	1.00	1.00	1.00	1.00
Primary	1.36* (1.00,1.84)	1.01 (0.92,1.10)	1.01 (0.93,1.11)	1.36* (1.00,1.84)	1.26 (0.92,1.72)	1.28 (0.94,1.75)
Secondary/higher	1.22 (0.84,1.77)	0.88** (0.80,0.97)	0.88* (0.80,0.97)	1.22 (0.84,1.77)	1.08 (0.74,1.59)	1.10 (0.75,1.62)
Unknown	1.09 (0.80,1.48)	1.09 (0.97,1.22)	1.09 (0.97,1.22)	1.09 (0.80,1.48)	1.19 (0.85,1.68)	1.21 (0.86,1.71)
<b>Exposure X household size</b>						
Father died/medium HH size			0.75 (0.28,2.00)			0.49 (0.13,1.87)
Father died/large HH size			0.63 (0.23,1.74)			0.80 (0.20,3.19)
Mother died/medium HH size			0.49* (0.26,0.91)			0.32* (0.13,0.79)
Mother died/large HH size			0.40* (0.20,0.82)			0.15*** (0.05,0.43)
Other adult died/medium HH size			0.78 (0.52,1.16)			1.04 (0.58,1.88)
Other adult died/large HH size			0.34*** (0.23,0.52)			0.64 (0.34,1.22)
<i>Constant</i>		0.13*** (0.12,0.15)	0.12*** (0.11,0.14)			
<i>Number of observations</i>		115575	115575		33328	33328
<i>Number of groups</i>		41305	41305		8656	8656
<i>Rho</i>		0.09 (0.08,0.11)	0.09 (0.08,0.11)			

\* $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$

Children resident in Viwandani slum had about 8% higher odds of changing residence than those from Korogocho. Children from the Luhya ethnic background had higher odds of changing residence while those from the Luo and Kamba ethnic backgrounds had lower odds as compared with their Kikuyu counterparts. Also, children from households with highest household wealth tertiles were significantly less likely to change residence than those from the poorest households. The effect was generally attenuated in the fixed effects model.

In the random effects but not the fixed effects model, children whose mothers had secondary or higher level education had lower odds of changing residence (OR=0.88,  $p$  value <0.01) than those whose mothers did not have any formal education. A child's gender and relationship to the household head were not significantly associated with higher or lower odds of a child changing place of residence.

In the random effects models, of the two outcomes examined here, *rho* (intra-cluster correlation coefficient) was small, meaning that the intra-cluster variability was large relative to the inter-cluster variability. To cross-check whether the sub-sample on which the outcome value varied over time was comparable to the total sample, random effects models were run on the sub-sample which had at least one outcome event happening and contributing information to the fixed effects model. The estimates were found to be comparable with those derived from the random effects model on the total sample meaning there was very little bias in the subsample. This observation suggests that there may be unmeasured confounders biasing the estimates from the random effects models which the fixed effects model controls for.

## 6.4 Discussion

While estimates from the different models for the two outcomes (outmigration and internal change of place of residence) are in same direction, their magnitudes differ. Based on the fact that the number of out-migrations and changes of residence events per individual is quite small, the estimates from the random effects model are likely to be more reliable and for this reason they are judged to better represent the relation between the exposure and outcome (outmigration) for this study. The conclusions and recommendations are thus based primarily on the results from the random effects

models. The results presented here confirmed that child mobility in this community is similarly high to that documented for the entire population in the slums <sup>[100, 133]</sup>.

Between 15-25% of children aged between 0-14 years out-migrate annually.

Additionally, a substantial proportion of children change place of residence within the slums in any given year. Proportions of children who out-migrated over the years were fairly comparable, with the exception of 2004, when there was marked increase. This peak corresponds to a government policy of clearing informal settlements built under or near high voltage power lines. Since no formal development can be authorised near or under high voltage power line, such areas often remain unoccupied and vulnerable to being encroached on by informal settlers. The government crackdown on informal structures constructed under the high voltage power lines peaked in 2004 but thereafter lost momentum due to weak enforcement but also due to public outcry that portrayed the exercise as inhumane and targeting the very vulnerable. The cleared areas have since been re-occupied by informal settlers. There was little fluctuation in proportion of children changing place of residence over the years. While every effort is made to verify out-migrations, it is possible that some children considered as out-migrants are just lost to the surveillance system but remain resident in same slum.

The migration experience of children who lived in households that experienced an adult death cannot be easily discerned from the descriptive results. Out-migration rates did not greatly vary by exposure status but more by slum of residence with the exception of calendar year 2007. A similar trend was observed with internal change of residence where only moderate differences were noticeable by exposure status but relatively big differences by slum of residence. The differences by slum confirm earlier findings that, while mobility is high in the two slums, it is much higher in Viwandani than Korogocho.

The author's expectation was that there would have been much higher proportions of child out-migration in exposed households as an adjustment to the adult death. This notion was based on previous observations elsewhere that showed that failing health, particularly of a household head mainly due to HIV/AIDS, increased the tendency to migrate away from the cities to rural homes <sup>[105]</sup>. As the results in earlier chapters showed, HIV/AIDS is the most important cause of adult death in this population and it

was therefore expected that there would be higher child out-migration from households with adult death than from other households. This was not the case except where a mother died. This finding may imply that with increasing availability of life improving and life prolonging highly active anti-retroviral therapy (HAART), the motivation for an ailing parent and their children to relocate to rural areas in anticipation of death might be less. HAART is more readily available in cities and this might motivate affected individuals to stay in the cities and probably remain in employment and continue looking after their family.

Looking at the proportions of internal changes of residence, relatively higher proportions are from households with an adult death. This link between adult death and intra-slum mobility suggests that affected children are more likely to be absorbed into existing households within the slums, rather than being sent away. Weak social links elsewhere, such as to their rural ancestral homes, may also act to limit out-migration. This observation is in line with an earlier study in South Africa that showed that adult death in a household was not associated with increased risk of household members out-migrating but rather an increased risk of households dissolving and its members being taken up into other households <sup>[24]</sup>. Another study in Tanzania also showed that death of a household head was associated with increased risk of household dissolution particularly of small households but did not identify the destinations of members of the dissolved households <sup>[110]</sup>. The findings in this study mean that generally children from affected households do not necessarily move out of the slums but rather move and join other households in the same community.

Results from the multivariable models showed that the risk of child out-migration depended on the child's relation to the deceased adult but also on the household size. Children whose mothers died were more likely to migrate out of the slums. This finding is in line with expectation. Mothers play a central role in child care and thus their death leaves a gap in the child care roles and as a consequence, affected children are likely to be moved to where relatives such as grandparents can provide day to day care, as has been the case with many HIV/AIDS orphaned children <sup>[25, 33, 284]</sup>. One study conducted in Malawi also found that maternal and double orphans were more likely to migrate <sup>[285]</sup>. That maternal orphans are more likely to out-migrate, presumably due to loss of care, is



supported by the observation that the likelihood of out-migration decreases with an increase in size of household, an indication that larger households have extra individuals to provide child care.

It is not clear why children who lived in households with a non-maternal adult death were less likely to out-migrate than children who lived in unaffected households. It could be that, in such households, the children's mothers are present and thus the choice is made to continue living with their children rather than sending them to live with other relatives. Results from the random and fixed effects models for death of a father and mother were generally comparable, but varied in magnitude of for the effect of a death of a non-parent adult. With regard to internal migration, the finding that children who lived in households that experienced an adult death were less likely to change place of residence was surprising. However, given that most adult deaths are due to HIV/AIDS, such deaths are predictable and anticipated and this may give time to households to plan placement of children in advance before the actual death occurs. As a result, it may appear as though death of an adult is followed by a reduced tendency of children to change place of abode.

The author's expectation was that children living in households headed by a non-prime age individual would be more likely to out-migrate due to difficulties in subsistence and child care. However, results showed that these children were less likely to out-migrate than others. Given that such children are not likely to be living with their parents, it might be that such children are already vulnerable and do not have many out-migration destination options. On the other hand, children in male-headed and non-parent headed households were more likely to out-migrate. This may point to lack of child care support in such households but also to the possibility that migration is a good thing in certain circumstances, as exhibited by higher streams among children from better-off households. For example, financially able parents may prefer sending their children away from slums to better residential schools.

Estimates from the random effects models showed that older children were less likely to out-migrate, which is in total contrast with estimates from the fixed effects model and expectation. This could be due to confounding from unmeasured variables in the

random effects model. The estimates and direction of association in the fixed effects model seem more plausible in this particular regard as older children need less care and can easily live with non-parents or go to school outside of the slums hence higher likelihood of out-migration. On the other hand, if the motivation to migrate is predominantly related to care and support, then older children are less likely to out-migrate. Unfortunately, the data used here do not allow the assessment of the motivation for migration. The observation that children from Viwandani slum were more likely to out-migrate can partly be explained by a higher tendency of adult migration in this slum than Korogocho. Additionally, there are differences in socio-economic status between the two slums which might be associated with migration but are not well controlled for by the wealth index used here. The foregoing observations imply that, while adverse events or circumstances might be a motivator (push) for out-migration, at the same time it appears that children from households with better social outlook have a higher tendency to out-migrate, probably seeking better opportunities, also referred to as pull factors. These contrast with push factors such as loss of income or care brought about by bereavement. For example, children whose mothers had better education and those from Viwandani, who can be assumed to have better socio-economic status, were more likely to out-migrate compared with those whose mothers had no education and their Korogocho counterparts.

Older children were less likely to change place of residence compared with those who lived in households headed by men or by non-prime age individuals. Older children tend to be in school and are thus less likely to move about as this would impact on their schooling. For those living in households with non-prime-age household heads, part of the reason for having a lower likelihood of changing residence might be related to having limited options given that they are already residing in less favourable arrangements. As expected, children in Viwandani were more likely to change place of abode and this might be for similar reasons as outlined for out-migration. The observation that children whose mothers had secondary or higher education were less likely to change residence might be related to financial security as seen in the results of household wealth as well as those for maternal education attainment.

## **Limitations**

While great efforts are made to track all registered members in the surveillance area, some individuals are lost to follow up and these might occasionally be misclassified as out-migrated. For children who out-migrate, the motivation and destination for migration is not known and this leaves a gap in our understanding of migration dynamics. In anticipated adult deaths, child outmigration might be planned to precede such death. If this happened, it might give an impression of low internal migration or out-migration rates post adult death and thus bias the estimates.

## **Conclusions**

There is high mobility of children in the two slums irrespective of whether there is an adult death or not. However, death of a mother significantly increases the risk of a child out-migrating from the slums and this is more so for small households. Based on this, it can be expected that the consequences of migration might vary and also be context specific. Future research should establish the reason and motivation for migration so that this is accounted for in the analysis.

It should be noted that the selective out-migration of children who lose a mother might leave behind a pool of paternal orphans whose circumstances are different from those of maternal orphans. This may have implications for assessments of other child outcomes in the remaining child population such as schooling and survival due to selection bias. This may result into biased estimates between orphans and non-orphans as the potentially more disadvantaged maternal orphans are excluded from the pool of orphans through selective out-migration.

## **7. Impact of adult death on children's living arrangements**

### **7.1 Introduction**

This chapter describes children's living arrangements in terms of household composition, including the number of individuals in a household and household headship. These aspects of household composition are further examined in relation to households' experience of adult death in order to assess how the death of an adult household member may impact the subsequent living arrangements of children. While a body of knowledge on children's living arrangements in sub-Saharan Africa exists, re-evaluating the relationship between adult death and children's living arrangements is pertinent owing to the growing burden of prime age deaths due to HIV/AIDS and its varying impact in different part of Africa <sup>[1, 16, 19, 71, 88, 146]</sup>. No research has been carried out in a slum setting where living arrangements differ for economic and social reasons even in the absence of excess adult mortality.

In sub-Saharan Africa, the extended family still plays an important role in child rearing <sup>[147]</sup>. This traditional structure is, however, waning and moving towards smaller nuclear families, especially in urban areas. This has implications for the available pool of relatives willing to take responsibility for non-biological children <sup>[34]</sup>. Urban poverty in sub-Saharan African cities, coupled with recent increase in prime-age mortality due to HIV/AIDS, brings to the fore the potential for negative impacts on child well-being <sup>[8, 36]</sup>. There is clear evidence of an increasing incidence and prevalence of orphanhood, with a corresponding increase in the proportions of children living in households headed by non-parents <sup>[16, 19]</sup>.

Household composition can be influenced by death of members, birth of new members, migration for various reasons including work, schooling, marital union formation, or dissolutions among other reasons. In sub-Saharan Africa, orphaned and non-orphaned children's living arrangements vary widely across the continent <sup>[19]</sup>. Often these arrangements may be influenced by economic challenges, parental death, and access to better schooling facilities, furthering family ties and mentorship <sup>[16, 130, 286]</sup>. This

therefore means that not all children who live with non-parents are doing so because of a crisis such as a parental death or other form of vulnerability. In fact, research has shown that in South Africa, non-resident parents especially fathers are common yet they often continue supporting their children who do not live with them <sup>[145]</sup>.

Why is it important to understand changes in children's living arrangements in the context of adult death? The need to know who the child lives with is informed by the desire to have a favourable environment for child growth and development such as that parents often provide. In the Kenyan context, there is a growing urban poor population who have limited access to means of livelihood, or to amenities such as health care, schools, and housing. Furthermore, due to the ever increasing number of orphans, concerns exist that the extended family system that absorbs most orphans has been stretched to the limit and is no longer adequate to cope with the burden of orphans <sup>[19]</sup>. In the two slums under study, average household size is small, typically less than 3 people per household, and most adults are in informal employment and thus have no formal social security in retirement or benefits in cases of untimely parental death.

Potential effects of changes in living arrangements include sibling separation, emotional distress, and compromised access to health services and education <sup>[147]</sup>. However, it is important to note that living with non-parents does not always imply worse health and social outcomes for children. Some children who do not live with their parents, including orphans, may live in wealthier households with better access to education and health care <sup>[130, 147, 150]</sup>. They may also get remittances from their non-resident parents if they are not orphaned. These dynamics might explain some findings that have shown that orphaned children do not have worse social and health outcomes compared with non-orphaned children <sup>[7, 287]</sup>.

Using longitudinal data, this chapter set out to identify changes in children's living arrangements associated with death of an adult. This analysis will add to the body of literature on children's living arrangement in several ways. Most of the available literature is based on cross-sectional surveys <sup>[19, 25]</sup>. Using longitudinal data permits understanding of the temporal ordering of events and, thus, may support causal inferences. Studies have also shown heterogeneity in children's living arrangements in

sub-Saharan Africa, and thus understanding local contexts is very important. Additionally, it is important to elucidate the impact of adult death on children's living arrangements in situations where coping strategies may be different as is likely to be the case with slums. The hypothesis being tested here is: adult death in a household influences potentially unfavourable children's living arrangements.

## **7.2 Methods**

### **Measurements**

#### **Living arrangements**

In this analysis, living arrangements refer to relationships, household headship and composition of household members. Generally, the concept encompasses relationships such as marital, parental and others in a household, household size, and household headship<sup>[288, 289]</sup>. Components of some of these are used in this study to represent/measure children's living arrangements.

#### **Household**

A household may be either: (a) "A one-person household, in which case, a person makes provision for his or her own food or other essentials for living without combining with any other person"; or (b) "A multi-person household, refers to a group of two or more persons living together and make common provision for food or other essentials for living. The persons in the group may pool their resources and have a common budget and may be related or not"<sup>[290]</sup>. A household head: "refers to that person in the household who is acknowledged as such by the other members"<sup>[288]</sup>. A child-headed household is one headed by an individual less than 15 years of age while elderly-headed households are those headed by an individual aged 60 years and above.

#### **Data preparation and analysis**

Descriptive statistics of children's living arrangements in the two slums are presented by calendar year. The living arrangements variables include the nature of household headship (gender, age), household size, and children's relationship to the household. These are estimated separately by child's exposure status. Age of household head and

household size are measured as means with their standard deviations while sex of household head and child's relationship to household head are presented as proportions.

In the multivariable analyses, random effects models were used to access the relationship between the key exposure variable (death of an adult) and the outcome variables. A choice between random effects models over fixed effects models was based on model comparison using Hausman test which tests the null hypothesis that the coefficients estimated by the more efficient random effects model are the same as those estimated by the consistent but less efficient fixed effects model. A significant result indicates that one should probably use a fixed effects model. However in this analysis, although the Hausman test was significant, the random effects model results have also been presented. This was done to obtain estimates for time-invariant covariates, which can only be produced by the random effects models. Also presentation of the two models helps to see if there was likely bias in the random effects estimates due to unmeasured confounders which is not an issue with the fixed effects models. With a few exceptions, the direction and magnitude of association from the two models on most covariates were similar.

For the multivariable analysis, the outcome variables examined included household head's age and household size as continuous variables as the rest of the variables examined in the descriptive analysis did not show significant relationship with the main exposure variable. Exposure status of a child was defined as death of one or more adults in a household where child lived. Control variables included household socio-economic status measured, child's sex, age, ethnicity, maternal education and slum of residence. Interaction terms that were significant according to the likelihood ratio test (exposure and household size) were also added to the models.

### **7.3 Results**

Table 22 shows descriptive results for children's living arrangements in households that did not experience an adult death, presented by year and by slum of residence. Aspects of living arrangements examined in the descriptive statistics included: household size, gender and age of household head, and relationship of the child to the household head.

In households that did not experience an adult death, the average household size did not vary much over the years and ranged between 3.7 persons per household for the years 2003, 2005 and 2006 to 3.8 persons per household in the years 2004 and 2007. This average is higher than the average household size for all households under surveillance including those with no children estimated at 2.5 persons per household. For all years examined, average household size for Korogocho slum was consistently larger (with the largest being 4.2 in 2004) than that for Viwandani slum (largest was 3.5 in 2004 and 2007). This observation is mirrored in the general slum population where household size for Korogocho is larger (2.9 persons per households) than that for Viwandani (2.3 persons per household). There seem to be no discernible trend in average household sizes over the period 2003 to 2007.

Most unexposed households were headed by men ranging from 79.1% in 2007 to 80.4% in 2004. Consistently, higher proportions of households were headed by men in Viwandani slum than Korogocho, ranging from 77.1% in 2004 in Korogocho to 84.3% in Viwandani slum in 2006. Generally, a higher proportion of children in Korogocho slum lived in households headed by women compared with Viwandani with the highest proportion being 24.6% in 2004 compared to 16.1% in Korogocho in 2003, 2004, 2005 and 2007. The average age for household heads was quite low, slightly varied by year, and ranged between 35.6 years in 2003 to 36.1 years in 2007. For all the years, mean ages for household heads were higher in Korogocho than Viwandani.

About 73% of children lived in households where the household head was the father. The range was quite narrow with lowest being 72.7% in 2005 and highest 73.4% in 2004.

A higher proportion of children in Viwandani than Korogocho lived in households where the household head was the father for all the years with a percentage difference of more than 10 points over the years. The proportions of households headed by a mother ranged between 14.3% in 2004 to 15.8% in 2007. Consistently, a higher proportion of children in Korogocho (ranging from 14.7% in 2007 to 15.6% in 2004) lived in households headed by a non-parent compared with Viwandani (ranging 8.2% in 2006 to 9.1% in 2003 and 2005).



Table 22: Living arrangements of children who lived in households that did not experience an adult death between 2003 and 2007

Variables	Year														
	2003			2004			2005			2006			2007		
	Korog-ocho	Viwan-dani	Total	Korog-ocho	Viwan-dani	Total	Korog-ocho	Viwan-dani	Total	Korog-ocho	Viwan-dani	Total	Korog-ocho	Viwan-dani	Total
Average HH* size	4.0	3.4	3.7	4.2	3.5	3.8	4.0	3.4	3.7	4.0	3.4	3.7	4.1	3.5	3.8
Sex of household head															
Female	23.8	16.1	20.1	22.9	16.1	19.7	24.2	16.0	20.5	24.6	15.8	20.5	25.4	16.1	20.9
Male	76.2	83.9	79.9	77.1	83.9	80.4	75.8	84.0	79.5	75.4	84.3	79.5	74.7	83.9	79.1
Mean age of HHH <sup>\$</sup> (SD)	37.3 (11.0)	33.6 (8.1)	35.6 (9.9)	37.5 (11.1)	33.6 (8.2)	35.7 (10.0)	37.4 (11.0)	34.0 (8.3)	35.9 (10.0)	37.3 (11.1)	34.2 (8.1)	35.9 (10.0)	37.7 (11.2)	34.5 (8.1)	36.1 (9.9)
Relation of HHH to child															
Father	68.0	78.5	73.0	68.2	79.1	73.4	67.3	79.2	72.7	67.0	79.6	72.9	66.6	79.0	72.6
Mother	17.0	12.4	14.8	16.3	12.1	14.3	17.6	11.7	14.9	18.1	12.2	15.4	18.7	12.6	15.8
Grandparent	5.1	2.1	3.7	5.5	2.2	3.9	5.5	2.4	4.1	5.4	2.1	3.9	5.5	2.1	3.9
Other	9.9	7.0	8.5	10.0	6.7	8.4	9.6	6.7	8.3	9.5	6.1	7.9	9.1	6.3	7.8
Number	10,501	9,621	20,122	11,793	10,763	22,556	11,867	9,757	21,624	11,997	10,327	22,324	12,017	11,043	23,060

\*Household; <sup>\$</sup> Household head

Table 23 shows descriptive results for children's living arrangements in households affected by an adult death by year and slum of residence. As for the unexposed households, the aspects of living arrangements presented here include average household size, gender of household head, mean age of household head and household head's relationship to the child.

In households that experienced an adult death, the average household size varied slightly between 2003 and 2007. There was a small declining trend from 4.5 in 2003 to 4.1 in 2007. On average Viwandani households were smaller than Korogocho with largest being 4.7 in Korogocho in 2003 and smallest being 3.5 in Viwandani in 2007. With the exception of 2007, most exposed households were headed by a man. There was a relatively clear decline in proportions of exposed households headed by women over the years. While only about 30% of exposed households were headed by a woman in 2003, this increased to 35% in 2004, 39% in 2005, 43% in 2006 and 52% in 2007. Overall, the proportion of households headed by women was lower in Viwandani compared with Korogocho.

Mean age of household heads for exposed households did not vary much over the years with highest average being 40.2 years in 2004 and 2005 and lowest being 39.7 years. Household heads in Viwandani slum were on average younger than their Korogocho counterparts with a difference ranging between 2 to 4 years.

Most exposed households were headed by a child's father. A higher proportion of households in Viwandani slum were headed by a child's father compared with Korogocho. The proportions of households headed by a father decreased over the years from 59% in 2003 to 53% in 2004, 51% in 2005, 47% in 2006 and 38% in 2007 with steeper decline in Viwandani slum. The proportion of households headed by a grandparent varied in a narrow range of 10.2% in 2003 to 12.3% in 2007 with small differences by slum of residence. For Korogocho slum, there seem to be a small but steady increase in proportion of households headed by a grandparent.

Table 23: Children's living arrangements for children who lived in households that experienced an adult death between 2003 and 2007

Variables	Year														
	2003			2004			2005			2006			2007		
	Koro-gocho	Viwa-ndani	Total	Koro-gocho	Viwa-ndani	Total	Koro-gocho	Viwa-ndani	Total	Koro-gocho	Viwa-ndani	Total	Koro-gocho	Viwa-ndani	Total
Average HH size	4.7	4.0	4.5	4.7	3.9	4.4	4.5	3.8	4.2	4.5	3.7	4.2	4.3	3.5	4.1
Sex of household head (HHH)															
Female	31.6	25.35	29.73	37.0	30.9	35.1	41.0	34.5	39.0	44.6	38.9	42.8	52.7	50.2	51.9
Male	68.4	74.65	70.27	63.0	69.1	64.9	59.0	65.5	61.0	55.4	61.1	57.2	47.4	49.8	48.1
Mean age of HHH	40.8	38.4	40.1	41.1	38.2	40.2	40.7	38.8	40.2	41	38	40.1	40.9	36.8	39.7
(SD)	(13.6)	(10.0)	(12.7)	(13.9)	(10.9)	(13.1)	(13.6)	(10.7)	(12.9)	(13.2)	(10.6)	(12.5)	(13.5)	(11.2)	(13.0)
HHH Relation to child															
Father	56.6	66.0	59.4	51.4	57.4	53.3	48.7	54.5	50.5	45.7	51.1	47.4	37.1	39.2	37.7
Mother	16.5	11.7	15.0	21.0	19.6	20.6	24.1	21.3	23.3	26.6	26.1	26.5	31.3	34.4	32.3
Grandparent	9.7	11.4	10.2	10.4	9.7	10.2	10.5	10.5	10.5	11.6	9.2	10.8	13.1	10.6	12.3
Other	17.3	10.9	15.4	17.1	13.3	15.9	16.7	13.8	15.8	16.1	13.6	15.4	18.5	15.8	17.7
Number	845	359	1,204	875	392	1,267	846	371	1,217	812	360	1,172	718	311	1,029

\*Household; \$ Household head

Figure 14 shows the proportion of children living in households headed by an individual aged less than 15 years or 60 years and above by exposure status. For all the years, a higher proportion of children who lived in exposed households lived with either a fellow child or an elderly person as their household head compared with those who lived in non-exposed household. For each of the years under observation the proportion of children living with non-prime age adults was more than double in exposed households compared with non-exposed households. Overall, there is no clear discernible trend in these proportions over time.

Figure 14: Proportion of exposed and non-exposed children living in households headed by either a child under 15 years of age or an elderly person of 60 years and above.

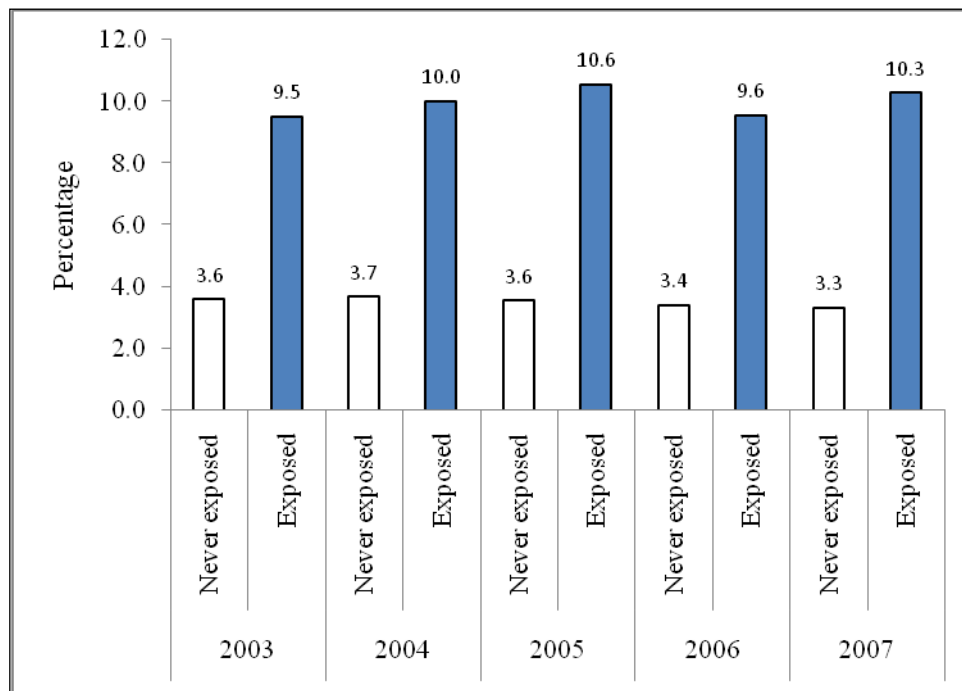


Table 24 shows results from random and fixed effects models of the association between household size and death of an adult in a household controlling for child's socio-demographic and household characteristics. Unadjusted and adjusted results are presented for both random and mixed effects model.

Results from the random effects model show that death of an adult in household was significantly associated with household size. In the adjusted models, it can be seen that death of a father was associated with an increase in household size while death of a mother or other adult was associated with a decrease in household size controlling for household size at the beginning of observation. In the fixed effects model, death of a father or non-parent adult was associated with an increase in size of the household a child used to live, while death of a mother was associated with a decrease in household size, though not significant.

In both adjusted and unadjusted random effects models, it can be seen that age of the household head was significantly associated with household size. Having non-prime age household head was associated with approximately 0.1 increase in the number of household members, implying that generally on average, households with older or child heads tend to have more household members, controlling for other factors. In the fixed effects model, direction of association was same as that from the random effects model but slightly different magnitude.

Households with non-parent household heads were generally larger than those where the household head was one of the parents. Non-parent headed households had on average 0.43 (95% CI: 0. 40, 0.45; p value<0.001) more members compared with those where the household head was a parent. The estimate was even more pronounced in fixed effects model. Results also showed that households headed by males were larger than those headed by females by about 0.36 members, holding all other variables constant.

Table 24: Factors associated with household size: Results from random and fixed effects models

Variables	Random effects models		Fixed effects models	
	Unadjusted $\beta$ coefficient (95% CI)	Adjusted $\beta$ coefficient (95% CI)	Unadjusted $\beta$ coefficient (95% CI)	Adjusted $\beta$ coefficient (95% CI)
Exposure				
Not exposed (Ref)				
Father died	1.35*** (1.24,1.46)	0.19*** (0.11,0.27)	1.34*** (1.20,1.48)	0.70*** (0.57,0.84)
Mother died	0.08 (-0.06,0.22)	-0.27*** (-0.35,-0.18)	-0.12 (-0.30,0.07)	-0.03 (-0.21,0.15)
Other adult died	0.69*** (0.61,0.77)	-0.14*** (-0.19,-0.09)	0.39*** (0.28,0.49)	0.44*** (0.34,0.54)
Household size at start	0.87*** (0.86,0.87)	0.82*** (0.81,0.82)		
Prime age HHH				
Prime age (Ref)				
Non-prime age	0.77*** (0.73, 0.81)	0.13*** (0.10,0.16)	0.31*** (0.26,0.35)	0.31*** (0.27,0.36)
Child's relation to HHH				
Parent (Ref)				
Non-parent	0.40*** (0.37,0.44)	0.43*** (0.40,0.45)	0.12*** (0.07,0.17)	0.80*** (0.74,0.86)
Sex of HHH				
Female (Ref)				
Male	0.82*** (0.79,0.85)	0.36*** (0.34,0.38)	1.04*** (1.00,1.08)	0.94*** (0.90,0.98)
Age category for child				
0-4 yrs (Ref)				
5-9 yrs	0.39*** (0.36,0.41)	0.10*** (0.08,0.12)	0.19*** (0.17,0.22)	0.22*** (0.19,0.24)
10-14 yrs	0.65*** (0.62,0.68)	0.15*** (0.13,0.17)	0.25*** (0.21,0.29)	0.32*** (0.28,0.36)
Sex of child				
Female (Ref)				
Male	-0.07*** (-0.11,-0.03)	-0.02** (-0.04,-0.01)		
Slum				
Korogocho (Ref)				
Viwandani	-0.91*** (-0.95,-0.87)	-0.23*** (-0.26,-0.20)		
Ethnicity				
Kikuyu (Ref)				
Luhya	0.31*** (0.26,0.37)	0.08*** (0.05,0.10)		
Luo	0.65*** (0.60,0.70)	0.12*** (0.10,0.15)		
Kamba	-0.27*** (-0.33,-0.22)	0.00 (-0.03,0.03)		
Other	0.99*** (0.94,1.05)	0.20*** (0.17,0.23)		
Wealth				
Poorest (Ref)				
Middle	-0.31*** (-0.35,-0.26)	0.06*** (0.04,0.09)	0.41*** (0.21,0.61)	0.13 (-0.06,0.32)
Wealthiest	0.48*** (0.44,0.52)	0.03* (0.01,0.05)	0.31*** (0.17,0.44)	0.41*** (0.28,0.54)
Maternal education				
None (Ref)				
Primary	-1.57*** (-1.63,-1.50)	-0.28*** (-0.31,-0.24)	-0.36*** (-0.49,-0.24)	-0.50*** (-0.62,-0.38)
Secondary/higher	-1.63*** (-1.70,-1.56)	-0.29*** (-0.33,-0.25)	0.35*** (0.20,0.49)	0.09 (-0.06,0.23)
Unknown	-2.57*** (-2.65,-2.50)	-0.68*** (-0.72,-0.63)	-2.07*** (-2.19,-1.94)	-2.67*** (-2.81,-2.54)
Constant		1.09*** (1.04,1.14)		4.39*** (4.24,4.53)
Number of observations		115,575		115,575
Number of groups		41,305		41,305
Rho		0.38		0.82

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ ; Ref-Reference category; HHH-Household head

Children aged 5 and above, on average, lived in larger households compared with those less than 5 years old. Children aged 5-9 years on average lived in households that were about 0.1 more members compared with children aged less than 5 years. A child's sex was very weakly associated the size of the household where they lived but generally boys were more likely to live in smaller households compared with girls.

As expected, Viwandani slum households were on average smaller than those of Korogocho slum by about 0.2 members, while household socio-economic status was very weakly associated with household size. By ethnicity, compared with the Kikuyu, children from all other identifiable ethnic backgrounds tended to live in larger households but the differences were generally small. In the random effects model, higher maternal education was associated with smaller household sizes. Children whose mothers had primary education or higher education lived in households that were on average 0.3 members smaller than those where the mother had no formal education. The results in the fixed effects model were mixed with primary maternal education being associated with 0.5 less members compared with no formal maternal education while secondary or higher maternal education was associated with about 0.1 more household members on average as compared with no formal maternal education.

Table 25 shows results from random and fixed effects models on the relationship between death of an adult in a household where children lived and the age of the household head. Generally, results from both random and fixed effects models are of slightly different magnitude and but same direction with very few exceptions.

Results from the random effects model show that death of a father was associated with a child living in a household with a household-head who was on average 3.7 years older than household heads in unexposed households. The relationship is more exaggerated if death occurred to a child's mother. Death of a mother was associated with a child subsequently living households with a head who was on average 4.4 years older than that of household heads in unaffected households. Results from the fixed effects model mirror those from the random effects model but with small differences in the magnitude of the association.

Holding all other variables constant, results show that larger households on average had older household heads compared with smaller households while households where the head was not a biological parent to a child tended to have older household heads by about 0.4 years compared with those where the household head was the biological

parent to the child. Male household heads were on average older than female heads by more than 3 years, holding other factors constant.

Compared with children from the Kikuyu ethnic background, children from all other identifiable ethnic backgrounds had younger household heads. Viwandani slum households with children under the age of 15 year tended to have younger household heads compared with those in Korogocho slum on average by about 3.5 years. Households in the middle wealth tertile had older household heads than the poorest households, while the least poor households had younger household heads than the poorest households. In the fixed effects model, wealthier households were headed on average by older individuals. Children whose mothers had primary or higher education tended to live in households with younger heads. Primary maternal education was associated with household heads aged about 5.2 years younger than if the mother had no formal education, while for mothers with secondary or higher education, household heads were approximately 4.2 years younger than those from households where the mother had no formal education.

Estimates from the models with interaction terms show that, in unexposed households, on average, households of 4-5 members had a household head who was about 2.6 years older while for households with 6 or more members, the household was older by about 5.5 years compared to households with 3 or less members. Similarly, in households where a mother or father died, the relationship with age of household head depended on household size. Larger households had the older household heads.



Table 25: Factors associated with household head age: Results from random and fixed effects models

Variables	Random effects models			Fixed effects models		
	Unadjusted $\beta$ coefficient (95% CI)	Adjusted $\beta$ coefficient (95% CI)	With exposure * HH size interaction	Unadjusted $\beta$ coefficient (95% CI)	Adjusted $\beta$ coefficient (95% CI)	With exposure * HH size interaction
Exposure						
Not exposed (Ref)						
Father died	6.81*** (6.33,7.29)	3.73*** (3.30,4.17)	3.09*** (2.06,4.12)	6.74*** (6.19,7.30)	2.78*** (2.29,3.26)	1.58** (0.51,2.66)
Mother died	5.12*** (4.50,5.73)	4.36*** (3.82,4.91)	4.74*** (3.83,5.64)	5.25*** (4.51,5.99)	4.30*** (3.66,4.95)	3.83*** (2.84,4.83)
Other adult died	4.04*** (3.69,4.39)	3.27*** (2.95,3.59)	0.96** (0.40,1.51)	3.30*** (2.88,3.73)	2.66*** (2.29,3.03)	0.09 (-0.50,0.68)
Household size						
$\leq 3$ (Ref)						
4-5	3.51*** (3.41,3.61)	2.63*** (2.53,2.73)	2.55*** (2.45,2.65)	3.18*** (3.07,3.30)	2.01*** (1.90,2.11)	1.90*** (1.80,2.01)
6+	7.23*** (7.11,7.35)	5.50*** (5.38,5.62)	5.43*** (5.31,5.55)	5.88*** (5.75,6.02)	3.91*** (3.78,4.04)	3.80*** (3.67,3.93)
Child's relation to HHH						
Parent (Ref)						
Non-parent	-0.98*** (-1.14,-0.81)	0.47*** (0.31,0.64)	0.47*** (0.30,0.63)	-3.07*** (-3.28,-2.87)	0.45*** (0.25,0.65)	0.44*** (0.24,0.64)
Sex of HHH						
Female (Ref)						
Male	3.60*** (3.47,3.74)	3.18*** (3.05,3.31)	3.18*** (3.06,3.31)	5.18*** (5.03,5.34)	4.52*** (4.37,4.66)	4.52*** (4.37,4.67)
Age category for child						
0-4 yrs (Ref)						
5-9 yrs	2.41*** (2.32,2.50)	1.40*** (1.31,1.49)	1.40*** (1.31,1.49)	1.75*** (1.64,1.85)	0.11* (0.00,0.21)	0.11* (0.00,0.21)
10-14 yrs	4.62*** (4.49,4.75)	2.91*** (2.77,3.04)	2.91*** (2.77,3.05)	3.35*** (3.19,3.52)	0.27** (0.10,0.44)	0.27** (0.10,0.44)
Sex of child						
Female (Ref)						
Male	-0.12 (-0.31,0.06)	0.03 (-0.14,0.19)	0.03 (-0.14,0.19)			
Slum						
Korogocho (Ref)						
Viwandani	-3.34*** (-3.52,-3.16)	-3.45*** (-3.71,-3.20)	-3.46*** (-3.71,-3.20)			
Ethnicity						
Kikuyu (Ref)						
Luhya	-0.44** (-0.73,-0.16)	-1.09*** (-1.35,-0.84)	-1.09*** (-1.35,-0.83)			
Luo	-1.06*** (-1.32,-0.79)	-2.60*** (-2.84,-2.36)	-2.59*** (-2.83,-2.35)			
Kamba	-1.95*** (-2.23,-1.68)	-0.69*** (-0.95,-0.43)	-0.69*** (-0.94,-0.43)			
Other	1.68*** (1.39,1.97)	-0.21 (-0.49,0.06)	-0.21 (-0.48,0.06)			

Wealth						
Poorest (Ref)						
Middle	-0.84*** (-1.07,-0.62)	0.54*** (0.31,0.77)	0.54*** (0.31,0.77)	3.97*** (3.16,4.77)	2.07*** (1.37,2.77)	2.04*** (1.35,2.74)
Wealthiest	1.24*** (1.04,1.44)	-0.64*** (-0.85,-0.43)	-0.65*** (-0.86,-0.43)	1.91*** (1.37,2.46)	2.17*** (1.70,2.63)	2.13*** (1.66,2.59)
Maternal education						
None (Ref)						
Primary	-6.99*** (-7.29,-6.69)	-5.16*** (-5.45,-4.87)	-5.18*** (-5.46,-4.89)	-4.36*** (-4.84,-3.88)	-4.17*** (-4.60,-3.73)	-4.19*** (-4.63,-3.75)
Secondary/higher	-6.39*** (-6.72,-6.05)	-4.19*** (-4.51,-3.88)	-4.21*** (-4.52,-3.89)	-1.29*** (-1.87,-0.72)	-1.52*** (-2.05,-0.99)	-1.52*** (-2.05,-0.99)
Unknown	-12.48*** (-12.82,-12.14)	-9.54*** (-9.88,-9.20)	-9.56*** (-9.90,-9.22)	-15.42*** (-15.91,-14.93)	-14.02*** (-14.50,-13.54)	-14.07*** (-14.55,-13.58)
Year						
2003 (Ref)						
2004	0.65*** (0.58,0.72)	0.40*** (0.34,0.47)	0.40*** (0.34,0.47)	0.71*** (0.64,0.78)	0.67*** (0.61,0.74)	0.68*** (0.61,0.74)
2005	1.19*** (1.11,1.26)	0.78*** (0.71,0.85)	0.78*** (0.71,0.86)	1.31*** (1.23,1.38)	1.33*** (1.26,1.40)	1.34*** (1.26,1.41)
2006	1.83*** (1.75,1.91)	1.25*** (1.18,1.33)	1.26*** (1.18,1.33)	2.02*** (1.94,2.10)	2.06*** (1.98,2.14)	2.07*** (1.99,2.15)
2007	2.46*** (2.38,2.54)	1.72*** (1.64,1.80)	1.73*** (1.65,1.81)	2.73*** (2.65,2.82)	2.82*** (2.73,2.91)	2.83*** (2.74,2.92)
<b>Exposure X household size</b>						
Father died /3-4 members			0.23 (-0.86,1.32)			0.72 (-0.39,1.83)
Father died/5+ members			0.82 (-0.32,1.97)			1.49* (0.31,2.67)
Mother died/3-4 members			-0.10 (-0.98,0.78)			0.75 (-0.15,1.65)
Mother died/5 + members			-1.08* (-2.09,-0.07)			0.06 (-0.97,1.10)
Other died/3-4 members			2.89*** (2.31,3.47)			3.17*** (2.58,3.75)
Other died/5 + members			2.82*** (2.20,3.43)			3.21*** (2.58,3.84)
Constant		35.58*** (35.20,35.96)	35.64*** (35.27,36.02)		31.19*** (30.66,31.71)	31.31*** (30.78,31.83)
Number of observations		115575	115575		115575	115575
Number of groups		41305	41305		41305	41305
Rho		0.85	0.86		0.89	0.91

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ ; Ref-Reference category; HHH-Household head

## 7.4 Discussion

The analysis in this chapter explored relationships between death of an adult in a household and children's living arrangements in the affected households. For the household size outcomes, the coefficients for the key exposure variable from the different models were of different magnitude but operated in the same direction. However, given that the fixed effects model did not control for the initial household size and yet this is important in determining/ influencing future household size, the discussion focuses on the results from the random effects model. The random and fixed effects models produced similar results for the household head's age.

The results showed that death of an adult in a household affected household size. Death of a father was associated with an overall increase in household size while death of a mother or other adult resulted in a decrease in household size. It might seem obvious and intuitive that death of a household member inevitably results into a decrease in household size by 1. Often, however, rearrangements in household membership occur either through other relatives moving in to fill the void or some members of the same household leaving the affected household to join or form other households within or outside of the slums. From these results, it is apparent that the motivation to change place of residence and hence household size of households where children live depends on how they are related to the deceased. The literature shows that children are less likely to live with their surviving father when their mother dies than they are to live with their mother if their father dies <sup>[16, 291]</sup>. Because widowed mothers tend to live with their children, chances are they bring on board other relatives to their households to help with child care and or income generation hence the increase in average household size for households where a father died. This might be in the form of a domestic servant, child's grandparent or a new spouse. On the contrary, if a mother dies, her children especially the very young ones, are more likely to be sent away to relatives who can provide child care such as grandparents or aunts even if their father remarried. If this happened, it would effectively reduce the household size. Also when a mother dies, there is a higher likelihood of her children under the age of 5 years dying and if this were to happen, the affected households would subsequently reduce in size following a mother's and her children's death with implications on the household and its surviving children if any.

Concerning death of other adults in the household such as grandparents, it is not clear why such deaths also result into a decrease in household size. It is however important to note that the association observed was weak. Indeed, in the fixed effects model, it was in the opposite direction. However, this model did not control for initial household size as this is a time invariant variable. Given that this category (non-parent adults) is mainly composed of grandparents who play a key role in child care, it might be that their death, like that of a mother, creates a void in the roles they play and this may result in changes in children's living arrangements and thus household size.

Male-headed and wealthier households were generally larger than female-headed households. This might be related to men's higher incomes and thus ability to fend for larger households. The fact that on average households with non-parent heads generally tended to be bigger than those where the household head was the parent of the child means that such households are likely recipients of children from elsewhere because of their relatively better socio-economic status and thus ability to look after non-biological children. The flipside is that non-parent household heads could be grandparents or other relatives to whom children resort to for care when their own parents die or migrate.

Households with older children (5 years or more) were larger than those with children less than 5 years of age. This can be explained by the fact that older children are more able to look after themselves in terms of personal care and thus it is easier and more practical for them to live with non-parents in an extended family system compared with children under-five years of age. There are other reasons why older children may live with non-parents including attending school away from home, helping with income generation and domestic work <sup>[130, 150]</sup>. While some literature highlight the plight of the orphaned girl child, results here showed negligible differences in living arrangements by child's gender, implying that there is no gender bias in child placement in the slums.

With some exceptions, most research has shown that orphanhood impacts children's living arrangements. Orphans generally tend to live in larger, female or elderly-headed households or those of lower social status <sup>[19, 25]</sup>. The results in this study showed that larger households were more likely to be headed by non-parent heads, or older individuals. Lower maternal education and residency in Korogocho slum were

associated with larger households. Seemingly contradictory, the results here showed that household size was positively associated with socio-economic status. This might mean that extra hands probably translate into better socio-economic status of the household but it might also be that such households are generally more attractive to and able to support extra members compared with poorer households. The measure of socio-economic status used here, however, is not very sensitive to recent changes in household economic circumstances and thus not a very good control in these models.

Children living in households that experienced an adult death were more likely to subsequently live in households with either an older or a child head of household. Similar results have been noted elsewhere. Hosegood et al., in a 2007 study, report that in South Africa, Tanzania and Malawi, the proportions of households headed by a children under 15 years of age was less than 1%, but that a much higher proportion of orphaned children lived in households where the head was 60 years or older. These findings are in line with expectation. Currently, a large proportion of deaths due to HIV/AIDS are prime-age thus, depriving households of their primary income earners<sup>[1, 8]</sup>. While the numbers were rather small to allow for stable estimates, it appears that the effect of an adult death in a household on subsequent household headship is graver in Viwandani slum compared with Korogocho, as evidenced by the larger coefficients in stratified analysis (results not shown). Because Viwandani households are generally smaller than in Korogocho, death of one prime-age adult, for example in a single-parent household, leaves the household with no other adult. This exposes the household to the risk of being headed by a child or young adults or elderly individuals who may not be economically capable of fending for the household with potential consequences of household members migrating or the entire household dissolving.

The economic situation of elderly people in Kenya is unlike that in other places such as South Africa where the elderly are entitled to a non-contributory pension<sup>[292]</sup>. Faced with a challenge of looking after orphans, such grants help to cover the financial burden of providing for grandchildren. The slums are typically a cash economy with most residents earning an income from temporary employment or petty trade. There is no formal social security for the vast majority of slum residents. Therefore, in the event of death of a bread winner, the effects are likely to be instantly felt, necessitating the

remaining household members, including children, to step up and take up work to earn income for the household or drop out of school. Because children and the elderly are unlikely to be as productive as a prime age adult would, their incomes are likely to be low hence increasing the vulnerability of the household and its members.

### **Limitations**

Some children under surveillance could not be linked to their parents because either their parents do not live in the surveillance area or they live in the surveillance area but in different households. The implication might be that the effects of orphanhood are underestimated for those children whose orphanhood status is not known. The surveillance system also does not ascertain survival status of non-resident parents. Similar challenges have been noted by similar studies in Tanzania, Malawi and South Africa <sup>[16]</sup>.

While it would have been interesting to isolate the role of HIV/AIDS deaths in the observed children's living arrangements, the small sample of events made it impossible to explore this. Besides, in a population with a generalised HIV epidemic and complex social relationships at household level, it is generally difficult to identify unaffected households who have not lost a relative to HIV/AIDS, thus making attribution of impact difficult <sup>[291]</sup>.

Many surveys have defined old age as 60 or more. Previous research in the slums has shown that the proportion of individuals aged 60 and above is quite small and indeed for this analysis, the numbers were too small to allow for stable estimates. Faced with similar constraints, some research reports have defined an elderly person as an individual aged 50 or more in the context of the developing world <sup>[293]</sup>. The downside to this, is the inability to directly compare findings with majority of other studies that have used 60 years as the cut-off age for identifying elderly individuals. This analysis maintained the widely-used age cut of 60.

### **Conclusions**

Death of adults in a household, particularly of parents, impacts children's living arrangements. With regard to household size, the impact depends on whether the

dead adult is a mother, father or other adult. Children who lost their mothers tend to live in relatively small households, while those who lost a father tend to live in relatively large households. On the other hand, death of any adult was associated with children living with older household heads. These potentially unfavourable living arrangements are likely to have social and health ramifications for the affected children. These are explored further in chapters 8 to 11.

## 8. All-cause child mortality and associated factors

### 8.1 Introduction:

Child health is high up on the international development agenda and this is reflected in one of the millennium development goals (MDG 4) which aims to reduce child mortality, with a target of reducing under-five mortality rate by two-thirds between 1990 and 2015 <sup>[152]</sup>. Although under-five mortality has been reported to be falling in many countries, including some in sub-Saharan Africa, the fall of less than 2% per year is not fast enough to meet the MDG 4 target <sup>[294, 295]</sup>. Moreover, in some countries under-five mortality rates remain high and there has been little or no progress in reducing it <sup>[296]</sup>. The slow progress in improving child health is partly attributable to HIV/AIDS, which is thought to be having a marked influence on child survival through the effects of vertical transmission from infected mothers and orphanhood <sup>[165, 297]</sup>.

Under-five mortality in Kenya fell from 115 per 1000 live births in 2003 to 74 in 2008/9 but prior to that fall it had experienced a stagnation <sup>[298]</sup> [12, 13, 298, 299]. In spite of the decline, child mortality still remains high and might be very high in disadvantaged rural and urban informal settlements <sup>[38]</sup>. The data needed for monitoring national child mortality trends have become more available than in the past through the regular demographic and health surveys carried out every five years in most countries in sub-Saharan Africa. However, data for local use, especially cause-specific data, are largely lacking. The usefulness of local data in planning health care delivery has been demonstrated, for example, in the Tanzanian Essential Health Interventions Project (TEHIP) which used such data to plan interventions and subsequently reduced child mortality by 40% in 5 years <sup>[300, 301]</sup>. The data routinely collected in health-care facilities are far from complete and there is also lack of capacity to use whatever is available. All-cause child mortality data are more available. However, the estimates are often based on survey data derived from birth histories for women aged 15-49 years that retrospectively ask about the number of children ever born and their survival. This means that children whose mothers have died are not included and yet they are likely to be at a higher risk of death themselves given the loss of nutrition and care offered by the mother. As a result of this, the mortality rates are likely to be underestimated.



Understanding the relationship between an adult death and child survival is important in this population for a number of reasons. In the past two decades, there has been markedly increased adult mortality due to HIV/AIDS <sup>[1, 8, 242]</sup>. Increasing numbers of parental deaths means increasing level of orphanhood and associated vulnerability, including reduced chances of survival, growth, nutrition, and schooling <sup>[6, 281, 302, 303]</sup>.

While interventions such as anti-retroviral therapy and prevention of mother-to-child transmission of HIV (PMTCT) have begun to take effect, community responses provided the first line of defence, particularly against the social effects such as orphanhood. There have been reports of resilient societal coping mechanisms directly related to child survival, for example orphans being taken up by relatives such as uncles, aunts and grandparents <sup>[304, 305]</sup>. Two challenges however remain. The care offered by relatives may be sub-optimal due to financial resource constraints, thereby compromising children's social and health outcomes. High incidence of HIV/AIDS associated adult mortality means that even the guardians may succumb to HIV/AIDS, further pushing the orphans to relatives who may be more distant or to non-relatives who might be less able to cater for the orphans or less concerned for their welfare. In the informal settlements, the households are small, typically less than 3 persons per household, and hence contain few relatives to turn to in times of crisis <sup>[229]</sup>. Unlike rural settings, where the extended family structure is more extensive, and thus can be easily tapped on in times of need, it is not known how the urban poor cope with a crisis and what effect failure to cope might have on the most vulnerable members.

Against this background, I hypothesise that while structural, environmental and socio-economic factors have a strong effect on child survival, it is likely that death of an adult in the informal settlements negatively impacts children further but this has not been explored before. Furthermore, it is not known whether death of non-parental adults in the household significantly impact child survival, given that non-parent adults might be carers of orphans owing to the growing problem of orphaned and vulnerable children in the region <sup>[306, 307]</sup>.

The specific aim of the analysis in this chapter is to explore the levels of child mortality and the effect of death of an adult on child survival.

## 8.2 Methods and data sources

### Data analysis

All individuals aged less than 5 years at any time during their residency in either of the two slums were identified. The cause of death data file was linked to the child file for analysis. The data file was prepared for survival analysis accordingly. Children who died during the follow up period were labelled with 1 and those censored were labelled with 0. Descriptive results are presented as age-specific death rates and crude death rates by some socio-demographic characteristics. Neonatal, infant and under-five mortality rates and Kaplan Meier survival curves are also presented. Mortality rates were estimated using standard life table procedures with age categorised as follows: less than 1 month; 1-2 months; 3-5 months; 6-11 months; 12-23 months and 23-59 months.

Exposure was defined as death of an adult in the household where the child was residing. Unexposed children were those children resident in households that did not experience an adult death for the period that they were under observation. Given the fact that the effects of social exposure such as death of a parent or other carer may begin manifesting before the actual death, the period of exposure was initially taken to be two years before death of an adult and two years after<sup>[17]</sup>. However, after preliminary analyses, it was noticed that excess mortality was concentrated in the 1 year before and after death of an adult. Based on this, the exposure period was revised accordingly and limited to 1 year before and after adult death. The survival time for children who were in households that experienced an adult death more than one year before or after an adult's death were thus re-categorised under the non-exposed category. To be able to investigate the risk of child death in relation to the timing of an adult death, child's survival time was split up with date of actual exposure (date of death of an adult) as the reference point. The unexposed children were grouped in one category. The exposed children's survival time was grouped as duration from the actual death of an adult i.e. 0-5 months and 6-11 months before and after exposure.

Part of the analysis in this chapter focuses on establishing factors associated with loss to follow-up (out-migration) and also checks to see whether those identified factors are associated with increased risk of death. To address this, I made comparisons of how those lost to follow-up differed from those who were present by various characteristics.

Those who were present were compared with those who died and those who had been lost to follow-up. Both descriptive and multivariable analyses using multinomial logistic regression techniques were carried out. The outcome variable (end of observation status) was trichotomous; i.e. current resident; died or out-migrated.

The next part of the analysis focused on assessing risk of child death by exposure status. I used multivariable analyses using Piecewise Exponential Regression adjusting for clustering of child deaths at household-level. The piecewise exponential model assumes exponential decline in mortality within age groups and allows for assessment of risk of death at various ages. Control variables included children's socio-demographic characteristics such as age, sex, ethnicity and slum of residence. Household-level variables, and those related to the deceased or other adults in household, included household wealth status, cause of death for the adult, child's relationship to the deceased adult, co-residence with parents, maternal education, and maternal age at time child was born. Where there was more than one adult death in a household, the cause of the earlier death was taken to be the cause of death the household was exposed to. Too few instances of multiple adult deaths in a single household occurred to merit a separate category (two or more deaths). Cause of adult death was categorised into HIV/AIDS/TB related, injury related and death due to causes other than HIV/AIDS/TB or injuries. Variables relating to either the household or the deceased were linked to the child file via a household ID. Co-residence was taken to be a child sleeping in same household with a given adult ("both parents", "mother only", "father only", and "neither of the parent") as at the last date of observation. The category of "mother only" was further split into two: "lived with mother who was the household head or lived with mother who was not the household head. Calendar year was also included as a variable since mortality can vary by season or calendar year. This was obtained by splitting child's survival time between the years 2003 to 2007.

### **8.3 Results**

A total of 732 children under the age of five years died between 2003 and 2007. Table 26 gives a breakdown of the under-five population according to residence status at the last day of observation (current resident; died or out-migrated) by socio-demographic

characteristics. A total of 26,345 children lived in the two slums at one time or another between 2003 and 2007.

Overall, 2.8% of all children ended up as deaths during the observation period. About 2.6% of children who were not exposed died, compared to 16% of those who lost a mother. As expected, the proportion of deaths was highest among the neonates at 36%. Children resident in Korogocho, the Luo, those who lived with father but no mother and those who were born to a mother aged 40 and above had higher proportions of deaths compared with the other categories. A higher proportion of children who had lost a mother or father out-migrated compared with those who did not lose any adult in the household. Generally, higher proportions of exposed children die and also tend to have higher proportions of out-migrations. With regard to age, the proportion of children who out-migrate increased with age. Viwandani slum had higher proportions (41%) of children who out-migrated compared to 32% in Korogocho slum, while children from the Kamba ethnic background had the highest proportion of out-migrations. Higher proportions of children from wealthier households, those who lived with none of their parents and those whose mothers had primary or higher education out-migrated as compared with their counterparts.

**Table 26: Last residence status of children less than 5 years of age by socio-demographic characteristics**

Variable	Currently resident N=15,937	Dead N=732	Out-migrated N=9,676	Total N=26,345
Death of adult (exposure)				
No adult death	60.8	2.6	36.6	25,588
Father died	46.8	7.8	45.4	268
Mother died	43.7	16.0	40.3	181
Other adult died	55.0	6.8	38.3	308
Age group of child				
<1month	48.4	36.0	15.7	370
1-2months	58.4	8.0	33.6	788
3-5months	49.6	6.5	44.0	1,554
6-11months	48.6	6.2	45.2	3,049
12-23months	49.0	2.7	48.3	5,141
24-59months	68.2	0.7	31.2	15,443
Sex				
Female	60.7	2.6	36.8	12,991
Male	60.3	3.0	36.7	13,354
Residence				
Korogocho	64.6	3.6	31.8	12,394
Viwandani	56.9	2.0	41.1	13,951
Ethnicity				
Kikuyu	68.1	2.4	29.6	6,808

Luhya	57.8	2.6	39.6	4,514
Luo	56.4	5.1	38.5	5,528
Kamba	53.1	1.8	45.1	5,591
Cushites	77.3	1.8	21.0	1,603
Other	59.6	1.7	38.7	2,301
Wealth tertiles				
Poorest	57.3	2.7	40.0	8,330
Second tertile	60.5	1.7	37.8	7,843
Wealthiest	63.8	3.7	32.5	9,894
Unknown	36.0	2.9	61.2	278
Calendar year				
2003	51.7	4.0	44.3	4,007
2004	42.8	2.6	54.6	5,244
2005	48.7	3.7	47.7	3,893
2006	51.1	4.3	44.6	3,682
2007	82.4	1.4	16.2	9,519
Co-residence with parents				
Both parents	60.4	2.7	37.0	20,876
Mother is household head	63.4	3.0	33.5	2,472
Mother not household head	61.8	2.7	35.5	915
Father only	66.8	4.9	28.2	627
None of parents	54.0	2.6	43.4	1,455
Maternal education				
None	78.6	2.9	18.5	1,344
Primary	59.5	2.9	37.6	16,994
Secondary/higher	60.6	2.5	36.9	6,374
Unknown	55.4	2.5	42.2	1,633
Maternal age at child birth				
<20yrs	58.4	3.0	38.6	7,193
20-29yrs	59.1	2.6	38.3	15,061
30-39yrs	71.8	3.0	25.2	3,137
40+yrs	71.8	5.7	22.5	315
Unknown	56.2	2.5	41.3	639
Total	60.5	2.8	36.7	100.0

Table 27 shows results from a multinomial logistic regression model with the outcome variable categories being: current resident; died; or out-migrated. Those who died or out-migrated are being compared with those who were current residents at the end of study period or when they attained the age of 5 years. Results show that children exposed to adult death had higher likelihood of dying than non-exposed children, the highest being associated with a maternal death. On the other hand, it can be seen that exposure to an adult death was also associated with higher risk of out-migration from the surveillance area and it appears that the odds of out-migration are particularly high if the father dies.

After the neonatal period, the risk of dying drastically reduces. It then plateaus through to the age of 11 months while that of out-migration increase after the neonatal period, plateau between 3 and 23 months and reduce thereafter. A child's sex was associated with neither increased risk of death nor out-migration. Being a resident in Viwandani

slum was associated with 36% higher risk of out-migration but not death. Children of the Luo ethnic background had more than double the risk of death and over 50% higher risk of out-migration compared with their Kikuyu counterparts. Calendar year 2004 was associated with 30% lower risk of death but over 40% higher risk of out-migration compared with 2003 while 2007 had both lower risk of death (89%) and out-migration (83%) compared with 2003. Children living with a father but no mother had lower risk of out-migration (38% lower), while those who lived with none of the parents had higher risk of out-migration (54% higher) compared with those who lived with both parents. Higher maternal education was not associated with significantly lower risk of child death but was associated with higher risk of out-migration.

**Table 27: Factors associated with death and migration, results from a multinomial logistic regression model**

Variables	Dead Vs Current resident	Out-migrated Vs Current resident
Death of adult (exposure)		
No adult death	1.00	1.00
Father	3.15*** (1.69,5.88)	2.20*** (1.62,3.00)
Mother	5.93*** (3.46,10.16)	1.59* (1.11,2.27)
Other	2.53** (1.46,4.41)	1.53** (1.16,2.01)
Age group		
<1month	1.00	1.00
1-2months	0.17*** (0.12,0.25)	1.72** (1.20,2.47)
3-5months	0.16*** (0.12,0.23)	2.56*** (1.83,3.60)
6-11months	0.16*** (0.12,0.21)	2.54*** (1.83,3.53)
12-23months	0.06*** (0.05,0.09)	2.58*** (1.86,3.56)
24-59months	0.01*** (0.01,0.01)	0.87 (0.63,1.20)
Sex		
Female	1.00	1.00
Male	1.16 (0.99,1.36)	0.98 (0.93,1.04)
Residence		
Korogocho		
Viwandani	1.08 (0.83,1.41)	1.36*** (1.24,1.50)
Ethnicity		
Kikuyu	1.00	1.00
Luhya	1.21 (0.93,1.58)	1.53*** (1.40,1.68)
Luo	2.05*** (1.63,2.56)	1.55*** (1.42,1.69)
Kamba	0.91 (0.69,1.20)	1.66*** (1.53,1.81)
Cushitic	0.56* (0.35,0.90)	0.82** (0.70,0.95)
Other	0.81 (0.55,1.18)	1.31*** (1.17,1.46)
Wealth tertiles		
Poorest	1.00	1.00
Second Tertile	0.62*** (0.48,0.81)	0.80*** (0.74,0.86)
Wealthiest	1.06 (0.84,1.33)	0.93 (0.85,1.01)
Unknown	1.47 (0.67,3.21)	2.49*** (1.88,3.29)
Calendar year		
2003	1.00	1.00
2004	0.70** (0.54,0.90)	1.43*** (1.31,1.56)
2005	0.83 (0.64,1.07)	1.10* (1.00,1.21)
2006	0.89 (0.70,1.15)	0.92 (0.83,1.01)
2007	0.11*** (0.08,0.14)	0.17*** (0.16,0.19)
Co-residence with parents		

Both parents	1.00		1.00	
Mother is household head	1.09	(0.83,1.44)	1.01	(0.91,1.11)
Mother not household head	0.93	(0.59,1.46)	0.93	(0.79,1.09)
Father only	0.99	(0.59,1.66)	0.62***	(0.50,0.77)
None of parents	1.21	(0.77,1.91)	1.54***	(1.32,1.81)
Maternal education				
None	1.00		1.00	
Primary	1.09	(0.73,1.63)	2.23***	(1.90,2.63)
Secondary/higher	1.11	(0.72,1.71)	2.13***	(1.80,2.53)
Unknown	1.05	(0.58,1.89)	3.00***	(2.42,3.70)
Maternal age at child birth				
<20yrs	1.15	(0.95,1.39)	1.06	(0.99,1.13)
20-29yrs	1.00		1.00	
30-39yrs	0.96	(0.74,1.24)	0.63***	(0.57,0.70)
40+yrs	1.77*	(1.01,3.12)	0.70*	(0.52,0.94)
Unknown	0.83	(0.36,1.90)	0.54***	(0.41,0.71)

The 732 child deaths that occurred between 2003 and 2007 accumulated 41,083 person-years of observation. Table 28 shows number of death, person-time and death rates by various characteristics. The overall crude death rate for children less than 5 years of age was 18 per 1000 person-years. The age standardised death rate among exposed children was on average more than twice that among unexposed children.

**Table 28: Death rates by exposure to adult death in household and other characteristics**

Variables	Person-years	Number of deaths	Crude death rate	ASDR (95% CI)
Death of adult (exposure)				
No adult death	39649	661	16.7	16.7 (15.5;17.9)
Father died	514	21	40.9	39.4 (23.0;55.7)
Mother died	323	29	89.5	83.3 (58.4;108.1)
Other adult died	597	21	35.2	34.6 (20.7;48.4)
Gender				
Female	20326	334	16.4	16.5 (14.8;18.2)
Male	20757	398	19.2	19.1 (17.3;20.9)
Residence				
Korogocho	20684	447	21.6	22.1 (20.1;24.0)
Viwandani	20399	285	14.0	13.7 (12.1;15.2)
Ethnicity				
Kikuyu	11512	162	14.1	14.3 (12.2;16.5)
Luhya	6633	119	17.9	18.2 (15.0;21.4)
Luo	8526	283	33.2	32.9 (29.3;36.6)
Kamba	7796	101	13.0	12.3 (9.9;14.6)
Cushitic	3138	28	8.9	9.3 (5.9;12.7)
Other	3480	39	11.2	11.4 (7.9;14.9)
Wealth tertiles				
Poorest	12331	227	18.4	18.3 (16.0;20.7)
Second tertile	12129	132	10.9	10.8 (9.0;12.6)
Wealthiest	16364	365	22.3	22.5 (20.3;24.8)
Unknown	259	8	30.9	29.3 (10.8;47.7)
Years				
2003	7764	160	20.6	20.5 (17.5;23.6)
2004	7762	136	17.5	17.2 (14.4;20.0)
2005	8219	144	17.5	17.5 (14.7;20.3)

2006	8631	159	18.4	18.7 (15.8;21.5)
2007	8707	133	15.3	15.4 (12.8;17.9)
Co-residence with parents				
Both parents	32895	563	17.1	16.8 (15.4;18.1)
Mother-Household head	3690	75	20.3	21.9 (17.1;26.6)
Mother-Not household head				
head	1542	25	16.2	16.3 (10.0;22.5)
Father only	1101	31	28.2	30.4 (20.3;40.5)
None of parents	1852	38	20.5	23.9 (16.6;31.3)
Education				
None	2818	39	13.8	16.8 (11.7;21.8)
Primary	26270	495	18.8	18.7 (17.1;20.3)
Secondary/higher	9941	158	15.9	15.5 (13.2;17.9)
Unknown	2053	40	19.5	19.5 (13.7;25.3)
Maternal age at child birth				
<20yrs	10684	212	19.8	23.2 (20.2;26.2)
20-29yrs	23511	393	16.7	16.0 (14.5;17.6)
30-39yrs	5557	93	16.7	15.7 (12.6;18.9)
40+yrs	569	18	31.6	27.7 (16.2;39.2)
Unknown	763	16	21.0	25.0 (13.3;36.7)
Total	41083	732	17.8(16.6;19.2)	

ASDR- Age standardised death rate

Mortality was higher among male children, residents of Korogocho, children born to mothers aged 40 years and above and the Luo ethnic group. Since 2003, there seems to have been a general downward trend in mortality among children though the confidence intervals for the different years overlap.

Table 29 shows the neonatal, infant and under-five mortality rates by some socio-demographic characteristics. Overall, the probability of a child dying before reaching their fifth birthday (U5MR) was 78 per 1000 live births. Infant mortality rate (IMR) was 50 per 1,000 live births. By slum of residence, Korogocho slum had a much higher IMR (59/1000) and U5MR (96/1,000) compared to Viwandani IMR (42/1,000) and U5MR (60/1,000). IMR (53/1,000) and U5MR (84/1,000) among males were higher than corresponding rates for females IMR (47/1,000) and U5MR (72/1,000).

Table 29: Under-five life table: Infant and under-five mortality rate by socio-demographic characteristics

Characteristic	Age group (Months)	Person-years	Deaths	Age-specific mortality rate	Cumulative probability of surviving	${}_1q_0^*$ & ${}_5q_0^s$ (per 1000 live births)
Sex						
Females						
	<1	379	66	0.174059	0.985631	
	1-2	776	26	0.033504	0.980147	
	3-5	1,176	50	0.042521	0.969800	
	6-11	2,318	82	0.035372	0.952843	47.2*
	12-23	4,353	67	0.015393	0.938323	
	24-59	11,324	43	0.003797	0.927714	72.3 <sup>s</sup>

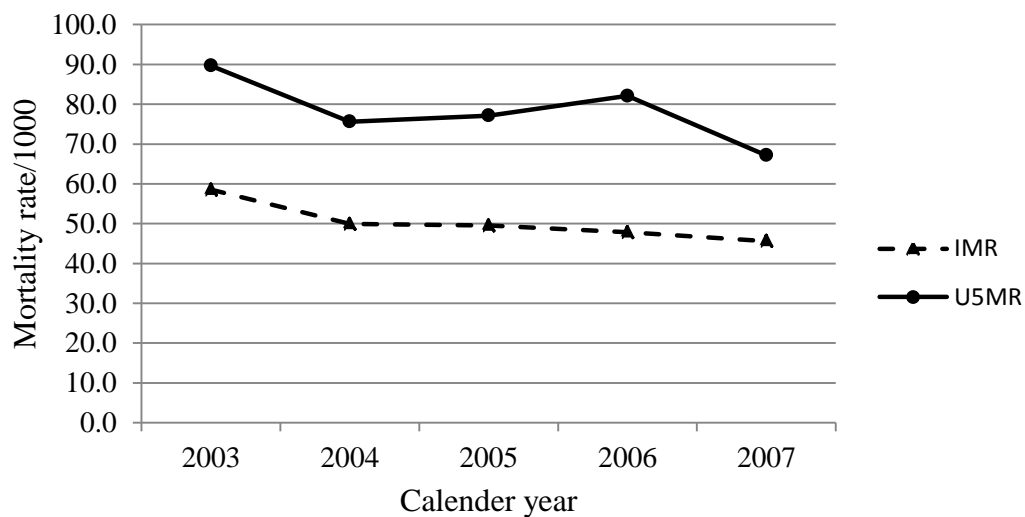


Males						
	<1	402	67	0.166621	0.986243	
	1-2	816	37	0.045364	0.978823	
	3-5	1,222	51	0.041730	0.968683	
	6-11	2,369	107	0.045175	0.947129	52.9*
	12-23	4,377	73	0.016677	0.931506	
	24-59	11,571	63	0.005445	0.916453	83.5 <sup>\$</sup>
Slum						
Korogocho						
	<1	381	76	0.199442	0.983517	
	1-2	773	40	0.051763	0.975069	
	3-5	1,162	66	0.056786	0.961323	
	6-11	2,294	99	0.043153	0.940803	59.2*
	12-23	4,327	92	0.021260	0.921012	
	24-59	11,747	74	0.006300	0.903769	96.2 <sup>\$</sup>
Viwandani						
	<1	400	57	0.142418	0.988202	
	1-2	819	23	0.028087	0.983587	
	3-5	1,236	35	0.028322	0.976647	
	6-11	2,393	90	0.037616	0.958449	41.6*
	12-23	4,402	48	0.010903	0.948056	
	24-59	11,149	32	0.002870	0.939927	60.1 <sup>\$</sup>
Overall						
	<1	781	133	0.170231	0.985914	
	1-2	1,592	63	0.039582	0.979432	
	3-5	2,398	101	0.042118	0.969173	
	6-11	4,687	189	0.040326	0.949826	50.2*
	12-23	8,730	140	0.016037	0.934716	
	24-59	22,895	106	0.004630	0.921822	78.2 <sup>\$</sup>

${}_1q_0^*$  - probability of dying between birth and exactly one year of age expressed per 1,000 live births

${}_5q_0^{\$}$  - probability of dying before reaching the age of five expressed per 1,000 live birth

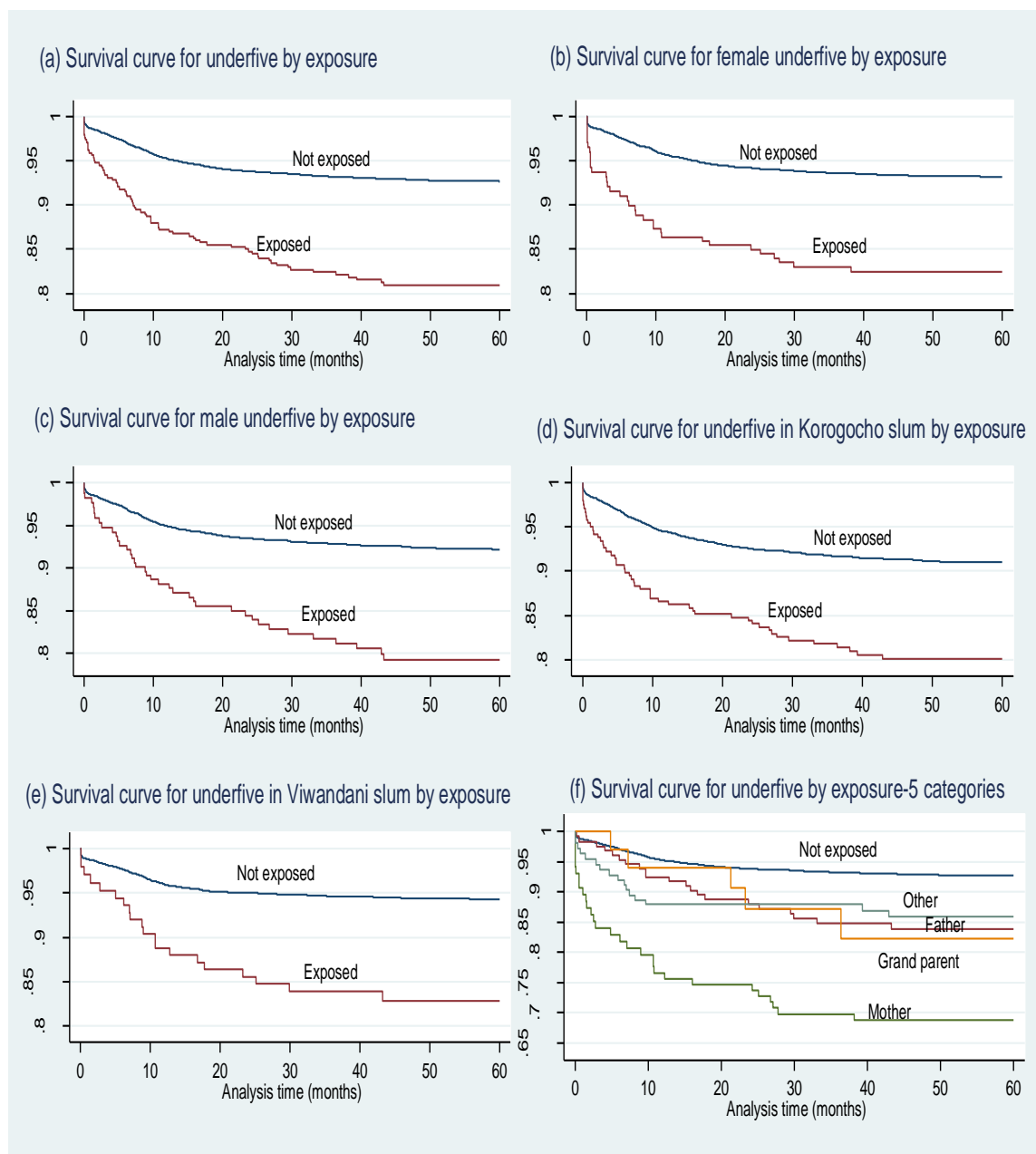
Figure 15: Infant and under-five mortality rate trends



For the convenience of presenting descriptive results, the exposed group was collapsed so that the exposure variable was a binary outcome i.e., exposed=1 and non-exposed=0

but is also presented as the five-category variable as earlier defined, Figure 16 (f). Figure 16 (a) shows survival curves by exposure status for all children, (b) survival curve by exposure status for females; (c) survival curve by exposure status for males; (d) survival curve by exposure status for resident of Korogocho slum (e) survival curve by exposure status for residents of Viwandani.

Figure 16: Survival curves for under-fives by exposure status



Log-rank test for equality of survivor functions for graphs (a) chi squared= 76.8; p value<0.0001; (b) chi squared=34.5; p value< 0.0001; (c) chi squared=42.8; p value< 0.0001; (d) chi squared=38.3; p value< 0.0001; (e) chi squared=28.5; p value< 0.0001 show that the exposed group had a lower probability of survival at all times under observation irrespective of their sex and slum of residence. Figure 16 (f) shows the survivor curve with the exposed category broken down to exposed-father died, mother

died or other adult died. The log-rank test for equality of survivor functions (chi squared= 110.1; p value<0.0001) indicate that the survival curves were significantly different. The risk of death was highest among children who lost their mother followed by those who either lost a grandparent, a father or other relative and lowest among the unexposed children.

Figure 17 shows survival curves by sex and slum of residence without taking into consideration exposure status. This was done to assess potential difference in the risk of death since other studies have shown that those variables may be important for child survival in their own right.

Figure 17: Survival curves for children aged less than 5 years by sex and slum of residence

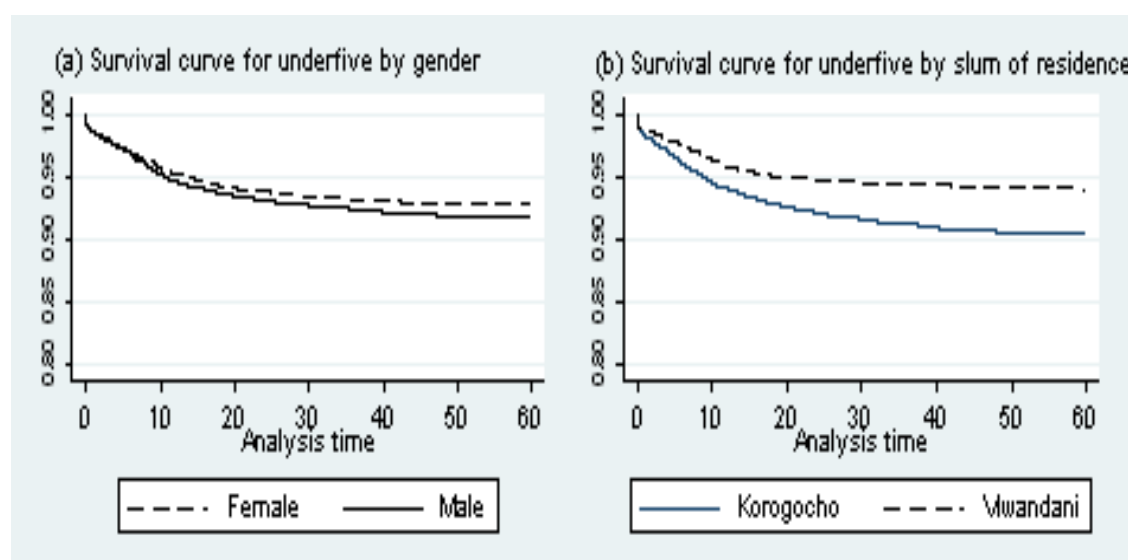


Figure 17 (a) shows survival curves by sex, (b) survival curves by slum of residence. The log-rank test for equality of survivor functions shows that survivor functions do not significantly differ by sex (a) -chi squared=3.8; p value=0.0515; while there was a significant difference in survivorship by slum of residence (b) -chi squared=41.38; p value<0.0001.

### **Risk of child death by exposure to an adult death**

Results presented in Table 30 provide an assessment of the relationship between adult death and survival of children in affected households. Model 1 has unadjusted hazard ratios of all the variables that were included in the final model (Model 6). The unadjusted results show that death of any adult is associated with at least double the risk of child death compared with children in households with no adult death. This risk was even larger if the dead adult was the child's mother (HR=5.4, p value <0.001). The results also show that the risk of death among exposed children was significantly elevated during the 12 months before and 6 months after an adult death. It appears that the most dangerous period is the 6 months after a mother's death. The risk to the child does not seem to differ by the cause of death of the adult as the confidence intervals for HIV/AIDS and TB, and injuries overlap. As expected, the risk of child death sharply decreases with child's age, particularly, after the first month of life. Boys were about 17% more likely to die than girls. Children resident in Viwandani were 35% less likely to die compared with those who were resident in Korogocho. Children from the Luo ethnic background were more than 2 times more likely to die compared with their Kikuyu counterparts while Cushites were about 37% less likely to die compared with the Kikuyu. Children from households classified as being in the middle tertile were about 41% less likely to die compared with the poorest. Although the risk of child death in all other calendar years in which children were observed was lower than that in 2003, only 2007 had significantly lower risk of death. Children residing with their father but no mother had a 64% higher risk of death compared with those who lived with both parents, while children who were born to mothers aged 40 years and above had 89% higher risk of death compared with those born to those in their twenties.

Model 2, 3 and 4 each examine a variable that represents a distinct aspect of exposure with the reference category being the un-exposed group controlling for age of the child. Model 2 looks at child's relationship to the dead adult categorised as "no dead adult" "father died" "mother died" "grandparent died" and "other adult died". The hazard ratios are all similar to those of the unadjusted model but slightly attenuated. Model 3 assesses child risk of death in relation to time of adult death with the categories of "no adult death" "0-5" "6-11" before adult death and "0-5" and "6-11" months after death. Exposures of more than 12 months were re-categorised as non-exposed. Controlling for

age, the hazard ratios were slightly attenuated compared with the unadjusted estimates. The third variable (model 4) focuses on cause of adult death categorised thus; no adult death, adult died of HIV/AIDS/TB, adult died of injuries and adult died of other causes. The hazard ratios remained similar to those of the unadjusted model.

Model 5 includes all three variables just discussed additionally controlling for age. Results show that death of a mother was associated with more than 3 times higher risk of death compared with children where there was no death of an adult in the household  $HR=3.14$ ,  $p$  value  $<0.001$ . The risk of death was highest in the first 6 months after death of an adult ( $HR=2.90$ ,  $p$  value  $<0.001$ ) while the hazard ratio for the 6 months prior to death of an adult was 2.29,  $p$  value  $<0.05$ . There were not significant differences in risk of child death by cause of adult death. The hazard ratios for age largely remained similar to those in the unadjusted model.

Model 6 is the full model with additional characteristics of the individual child and household. The results show that death of a mother was associated with 2.9 times higher risk of death of a child compared with children who lived in households that did not experience an adult death ( $HR=2.9$ ,  $p$  value  $<0.001$ ). The period 6 months before death of adult was associated with a 2.3 times higher risk while the following 6 months was associated with a 3 times higher risk compared with those who were not exposed. Cause of death of an adult was not associated with differences in the risk of death of children. Risk of death of children was highest among the neonates and gradually diminished by the age of 5 years.

Table 30: Risk of dying between 0 and 5 years: Regression results from piecewise exponential models

Variables	Model 1 (unadjusted)	Model 2	Model 3	Model 4	Model 5	Model 6
Relationship of dead adult to child						
Not exposed	1.00	1.00			1.00	1.00
Father died	2.45*** (1.61,3.73)	2.34*** (1.55,3.52)			1.31 (0.68,2.54)	1.03 (0.49,2.17)
Mother died	5.40*** (3.54,8.23)	5.19*** (3.49,7.72)			3.14*** (1.83,5.39)	2.89*** (1.67,5.01)
Others adult died	2.11** (1.34,3.32)	2.02** (1.30,3.15)			1.16 (0.60,2.26)	0.92 (0.46,1.83)
Period in which adult death occurred						
Not exposed or >12months	1.00		1.00		1.00	1.00
6-12 months before exposure	2.97** (1.55,5.68)		2.65** (1.39,5.06)		1.44 (0.67,3.07)	1.44 (0.67,3.09)
0-5 months before exposure	4.40*** (2.72,7.11)		4.37*** (2.70,7.07)		2.28* (1.20,4.35)	2.30* (1.20,4.39)
0-5 months after exposure	6.89*** (4.43,10.73)		5.09*** (3.25,7.97)		2.90*** (1.57,5.37)	3.03*** (1.62,5.66)
6-11 months after exposure	0.41 (0.06,2.91)		0.40 (0.06,2.88)		0.23 (0.03,1.72)	0.25 (0.03,1.89)
Cause of death of adult						
Not exposed	1.00			1.00	1.00	1.00 (1.00,1.00)
HIV/AIDS/TB	3.05*** (2.01,4.61)			3.08*** (2.07,4.59)	1.25 (0.73,2.11)	1.04 (0.61,1.78)
Injuries	3.02** (1.59,5.74)			2.82** (1.51,5.26)	1.36 (0.63,2.92)	1.37 (0.63,2.99)
Other causes	2.90*** (2.03,4.15)			2.70*** (1.91,3.82)	1.00 (1.00,1.00)	1.00 (1.00,1.00)
Child age group						
< 1 month	1.00	1.00	1.00	1.00	1.00	1.00
1-2 months	0.23*** (0.17,0.32)	0.23*** (0.17,0.32)	0.23*** (0.17,0.32)	0.23*** (0.17,0.32)	0.23*** (0.17,0.32)	0.23*** (0.17,0.32)
3-5 months	0.25*** (0.19,0.32)	0.25*** (0.19,0.32)	0.25*** (0.19,0.32)	0.25*** (0.19,0.32)	0.25*** (0.19,0.32)	0.25*** (0.19,0.32)
6-11 months	0.24*** (0.19,0.30)	0.24*** (0.19,0.30)	0.24*** (0.20,0.31)	0.24*** (0.19,0.30)	0.25*** (0.20,0.31)	0.25*** (0.20,0.31)
12-23 months	0.09*** (0.07,0.12)	0.09*** (0.07,0.12)	0.10*** (0.08,0.12)	0.09*** (0.07,0.12)	0.10*** (0.08,0.12)	0.10*** (0.08,0.12)
24-59 months	0.03*** (0.02,0.04)	0.03*** (0.02,0.04)	0.03*** (0.02,0.04)	0.03*** (0.02,0.04)	0.03*** (0.02,0.04)	0.03*** (0.02,0.04)
Gender (child)						
Females	1.00					1.00
Males	1.17* (1.00,1.36)					1.19* (1.02,1.37)
Slum of residence						
Korogocho	1.00					1.00
Viwandani	0.65*** (0.55,0.75)					1.07 (0.84,1.37)
Ethnicity						
Kikuyu	1.00					1.00
Luhya	1.28 (1.00,1.63)					1.29* (1.01,1.64)

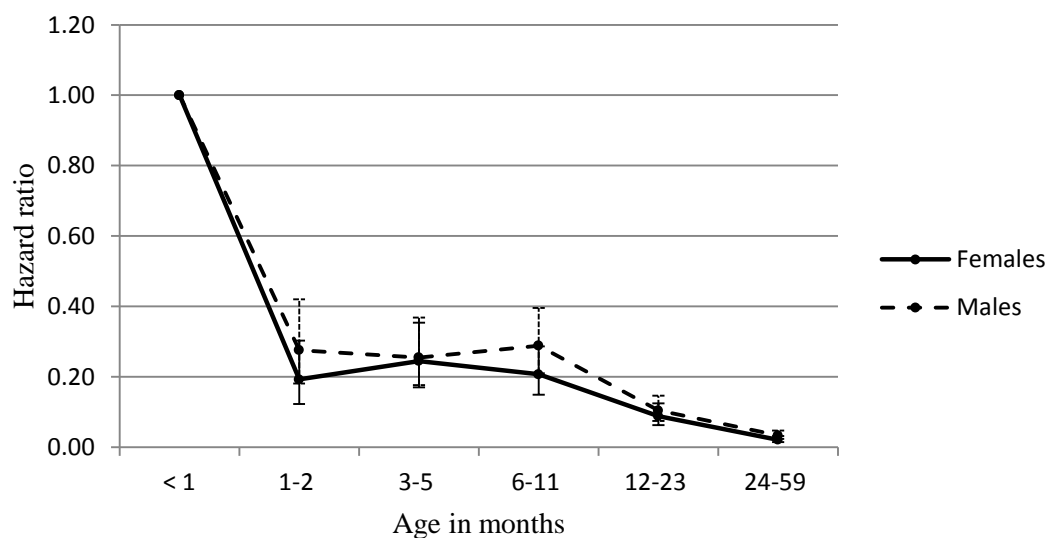
Luo	2.36*** (1.93,2.88)	2.05*** (1.67,2.50)
Kamba	0.92 (0.72,1.18)	0.96 (0.74,1.25)
Cushitic	0.63* (0.42,0.97)	0.60* (0.39,0.94)
Other	0.80 (0.56,1.13)	0.88 (0.61,1.26)
Wealth status		
Poorest	1.00	1.00
Middle	0.59*** (0.47,0.74)	0.63*** (0.50,0.81)
Wealthiest	1.21* (1.02,1.44)	1.07 (0.87,1.31)
Unknown	1.68 (0.85,3.35)	1.45 (0.71,2.93)
Calendar years		
2003	1.00	1.00
2004	0.85 (0.68,1.07)	0.84 (0.66,1.05)
2005	0.85 (0.68,1.07)	0.87 (0.69,1.09)
2006	0.89 (0.72,1.11)	0.95 (0.76,1.18)
2007	0.74* (0.59,0.93)	0.81 (0.64,1.02)
Co-residence with parents		
Both parents	1.00	1.00
Mother-Household head	1.19 (0.93,1.51)	1.26 (0.98,1.61)
Mother -not household head	0.95 (0.62,1.44)	0.92 (0.61,1.40)
Father	1.64** (1.15,2.35)	1.24 (0.80,1.92)
None of the parents	1.20 (0.86,1.67)	1.19 (0.77,1.83)
Maternal education		
No education	1.00	1.00
Primary	1.36 (0.97,1.91)	0.93 (0.65,1.32)
Secondary or higher	1.15 (0.80,1.65)	0.89 (0.61,1.30)
Unknown	1.41 (0.90,2.20)	0.88 (0.53,1.48)
Maternal age at child birth		
< 20yrs	1.19 (1.00,1.41)	1.23* (1.03,1.46)
20-29yrs	1.00	1.00
30-39yrs	1.00 (0.79,1.26)	0.93 (0.74,1.17)
40+yrs	1.89* (1.11,3.24)	1.62 (0.94,2.79)
Unknown	1.26 (0.76,2.08)	1.29 (0.60,2.77)

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$



Figure 18 shows the risk of death by sex at various age categories relative to the first month of life controlling for covariates. For both sexes, risk of death drastically reduces in the first month, plateaus between 1 and 11 months and then reduces gradually thereafter.

Figure 18: Risk of dying at various ages relative to the first month of life by sex after controlling for other covariates



Overall, male children had a 19% higher risk of death compared with female children. After controlling for covariates, slum of residency was not associated with differing risks of child death. By ethnic background, children from the Luhya ethnic background were about 29% more likely to die than the Kikuyu, while the Luo children were more than twice as likely to die than the Kikuyu (HR=2.05, p value <0.001). Children from the Cushites community were 40% less likely to die than children from the Kikuyu ethnic group. Children from households categorised as of middle wealth status were 37% less likely to die compared with children from the poorest households while those from the wealthiest were not significantly different from the poorest. Although co-residency with only father was associated with increased risk of death before controlling for other variables, this became non-significant upon controlling for covariates. Children who were born to teenage mothers were about 23% more likely to die compared with those born to mothers in their twenties. Interaction terms of exposure

and age group, sex, slum of residence, and ethnicity were tested but none were significant.

To facilitate understanding of whether there are differences in the risk of child death jointly by the child's relationship to the adult who died in household and what they died of, the exposure variable was redefined as; not exposed, father died of HIV/AID/TB, father died of injuries, father died of other cause, mother died of HIV/AIDS/TB, mother died of injuries, mother died of other causes, "other adult" died of HIV/AIDS/TB, "other adult" died of injuries and "other adult" died of other causes as shown in Table 31. The category "mother died of injuries" had few cases and was collapsed to "mother died of other causes".

The model building approach used in Table 30 was also used here. Model 1 has the unadjusted hazard ratios. The unadjusted estimates show that with the exception of "other adult died of other causes", the rest of the categories had higher risks of child death compared with the unexposed group. However upon controlling for all other covariates (Model 4), the only category that has significantly higher risk of death was that of children who lost a mother. The risk is highest (HR4.6, p value<0.001) if the mother died of HIV/AIDS/TB but also substantially high (HR=2.3, p value<0.01) if the mother died of causes other than HIV/AIDS/TB. It is important to note that although the estimates noted above look substantially different, their confidence intervals overlap. The rest of estimates for the covariates in the models remained very similar to those observed in model 5 in Table 30.

Table 31: Risk of dying between 0 and 5 years: Regression results from piecewise exponential models with exposure redefined

Variables	Model 1 (Unadjusted)	Model 2	Model 3	Model 4
Relationship & CoD of adult				
Not exposed	1.00	1.00	1.00	1.00
Father died of HIV/AIDS	2.92**(1.53,5.58)	2.89*** (1.57,5.33)	1.88 (0.91,3.92)	1.09 (0.47,2.48)
Father died of Injuries	2.82*(1.21,6.54)	2.43* (1.05,5.59)	1.66 (0.66,4.14)	1.49 (0.56,3.92)
Father died of other CoD	1.89(0.92,3.86)	1.84 (0.90,3.73)	1.21 (0.52,2.78)	1.00 (0.41,2.41)
Mother died of HIV/AIDS	9.16*** (4.94,16.98)	9.18*** (5.23,16.10)	5.68*** (2.95,10.94)	4.61*** (2.39,8.91)
Mother died of other CoD	3.90*** (2.20,6.93)	3.69*** (2.14,6.36)	2.53** (1.35,4.77)	2.33** (1.23,4.42)
Other adult died of HIV/AIDS	0.94(0.35,2.56)	0.97 (0.36,2.61)	0.63 (0.22,1.82)	0.46 (0.16,1.35)
Other adult died of Injuries	4.34** (1.65,11.40)	4.46** (1.79,11.08)	3.25* (1.28,8.25)	2.20 (0.80,6.03)
Other adult died of other CoD	2.72** (1.53,4.82)	2.43** (1.39,4.25)	1.58 (0.80,3.12)	1.23 (0.61,2.50)
Period in relation to time of adult death				
Not exposed or >12months	1.00		1.00	1.00
6-12 months before exposure	2.97** (1.55,5.68)		1.43 (0.67,3.09)	1.41 (0.66,3.03)
0-5 months before exposure	4.40*** (2.72,7.11)		2.30* (1.20,4.43)	2.22* (1.15,4.28)
0-5 months after exposure	6.89*** (4.43,10.73)		2.79** (1.51,5.14)	2.90*** (1.56,5.41)
6-11 months after exposure	0.41(0.06,2.91)		0.22 (0.03,1.66)	0.24 (0.03,1.82)
Child age group				
< 1 month	1.00	1.00	1.00	1.00
1-2 months	0.23*** (0.17,0.32)	0.23*** (0.17,0.32)	0.23*** (0.17,0.32)	0.23*** (0.17,0.32)
3-5 months	0.25*** (0.19,0.32)	0.25*** (0.19,0.32)	0.25*** (0.19,0.32)	0.25*** (0.19,0.32)
6-11 months	0.24*** (0.19,0.30)	0.24*** (0.19,0.30)	0.25*** (0.20,0.31)	0.25*** (0.20,0.31)
12-23 months	0.09*** (0.07,0.12)	0.09*** (0.07,0.12)	0.10*** (0.08,0.12)	0.10*** (0.08,0.12)
24-59 months	0.03*** (0.02,0.04)	0.03*** (0.02,0.04)	0.03*** (0.02,0.04)	0.03*** (0.02,0.04)
Gender (child)				
Females	1.00			1.00
Males	1.17*(1.00,1.36)			1.19* (1.02,1.38)
Slum of residence				
Korogocho	1.00			1.00
Viwandani	0.65*** (0.55,0.75)			1.07 (0.84,1.37)
Ethnicity				

Kikuyu	1.00	1.00
Luhya	1.28(1.00,1.63)	1.29* (1.01,1.65)
Luo	2.36*** (1.93,2.88)	2.05*** (1.67,2.51)
Kamba	0.92(0.72,1.18)	0.96 (0.74,1.24)
Cushitic	0.63*(0.42,0.97)	0.61* (0.39,0.95)
Other	0.80(0.56,1.13)	0.88 (0.61,1.26)
Wealth status		
Poorest	1.00	1.00
Middle	0.59*** (0.47,0.74)	0.64*** (0.50,0.81)
Wealthiest	1.21*(1.02,1.44)	1.06 (0.86,1.31)
Unknown	1.68(0.85,3.35)	1.44 (0.71,2.93)
Calendar years		
2003	1.00	1.00
2004	0.85(0.68,1.07)	0.84 (0.67,1.06)
2005	0.85(0.68,1.07)	0.87 (0.69,1.09)
2006	0.89(0.72,1.11)	0.95 (0.76,1.18)
2007	0.74*(0.59,0.93)	0.81 (0.64,1.02)
Co-residence with parents		
Both parents	1.00	1.00
Mother-Household head	1.19(0.93,1.51)	1.24 (0.97,1.59)
Mother -not household head	0.95(0.62,1.44)	0.93 (0.61,1.41)
Father	1.64** (1.15,2.35)	1.27 (0.81,1.97)
None of the parents	1.20(0.86,1.67)	1.17 (0.75,1.81)
Maternal education		
No education	1.00	1.00
Primary	1.36(0.97,1.91)	0.94 (0.66,1.36)
Secondary or higher	1.15(0.80,1.65)	0.89 (0.61,1.31)
Unknown	1.41(0.90,2.20)	0.86 (0.52,1.45)
Maternal age at child birth		
< 20yrs	1.19(1.00,1.41)	1.22* (1.02,1.45)
20-29yrs	1.00	1.00
30-39yrs	1.00(0.79,1.26)	0.93 (0.73,1.17)
40+yrs	1.89*(1.11,3.24)	1.65 (0.96,2.84)
Unknown	1.26(0.76,2.08)	1.35 (0.63,2.92)

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$

## 8.4 Discussion

### Mortality rates

Globally, the total number of under-five deaths decreased by about 28% between 1990 and 2008 from 12.5 million to 8.8 million <sup>[295]</sup>. While a lot has been studied concerning risk factors for child mortality, the stalling in the decline of child mortality in Kenya in the recent years largely remains unexplained. HIV/AIDS is often posited as the main reason for the stalling in the decline in child mortality based on evidence from elsewhere <sup>[165]</sup>. There have been no prospective studies that have examined how effects of HIV/AIDS interact with individual child, household and community level factors to influence child survival in the current circumstances. Related to this is the lack of clarity as to whether the high risk of child mortality is predominantly due to paediatric HIV infections or largely a consequence of losing parents and carers due to HIV/AIDS. This chapter explored mortality of children less than five years of age and also assessed the impact of death of an adult on child survival controlling for individual and household-level factors.

The results show that infant and under-five mortality rates in the two slums are high, though comparable to the national estimate <sup>[12, 13]</sup>. Neonatal mortality rate was 14 deaths per 1000 live births, infant mortality was 50 deaths per 1000 live births and under-five mortality rate was 78 deaths per 1,000 live births. According to the Kenya Demographic and Health Survey (KDHS) results for 2008/9, the neonatal, infant and under-five mortality rate for Kenya over the period 2004-2008 were 31, 52 and 74 deaths per 1000 live deaths respectively. The estimates of neonatal, infant and under-five mortality rate for Nairobi province, where the two slums are located, were 48, 60 and 64 deaths per 1000 live deaths respectively while the estimate for urban areas of the country were 32, 63 and 74 deaths per 1000 live births <sup>[13]</sup>. Thus, for neonatal and infant mortality rates, the estimates in this study are lower than those in the KDHS 2008/9 for Kenya as a whole and for the urban population. On the other hand, under-five mortality rate was higher in this study than that reported in the KDHS 2008/9. The estimates also mean that in the slums, the risk of child death remains high after 1 year of age hence the higher under-five mortality rate. This may be an indication that factors not related to childbirth such as household and environmental factors are important determinants of child survival in this community. The low neonatal mortality rate in the slums is hard to

explain. It is unlikely that mothers in the slums get better obstetric care and better newborn care compared with other parts of Nairobi province even though the slums are located close to the city's main health care facilities. Previous research has revealed poor access and utilisation of maternity and child care services by slum residents <sup>[308]</sup>. This leaves us with a possibility that neonatal infant deaths are generally under-reported. If this was the case, the explanation might be a tendency for pregnant women to be lost to follow-up, especially single mothers who might seek social support from their rural kin and may not return to the same slum after giving birth. If those who migrate and are lost to follow-up are more likely to lose their babies, then the resultant neonatal mortality might be underestimated. In fact, the results showed that children who lose their mother are not only more likely to be lost to follow-up but also they were more likely to die. Circular migration has been documented in this population and is reported to be highest among women. Further investigation to find out whether there is maternity related migration and how that might impact on child mortality estimates for the slums is needed <sup>[100]</sup>.

### **Risk of death following an adult death in household**

Results in this study showed that death of a mother, but not that of other adults in the household, increases the risk of child death by almost three-fold compared with children living in households with no adult death. This estimate was arrived at after controlling for known confounders and other known risk factors. This finding is not entirely novel, however it gives insights into the implications of high adult mortality, especially that of women, in the era of HIV/AIDS. Similar to this study, a recent study in Bangladesh showed that death of a mother in the neonatal period increased the risk of child death by over 8 times while that of a father had negligible effect <sup>[173]</sup>. Findings in this study contrasts with those published on a rural Malawian population where it was noted that death of a mother did not increase the risk of child death but rather the mother's positive HIV status did <sup>[6]</sup>. This seems to suggest that paediatric HIV infection rather than effects of orphanhood played a major role in child survival. It might also be a pointer to possible better orphan care in the rural Malawi context due to stronger kinship relations as compared with the slums of Nairobi. But it is important to note that the Malawi study took place in a period before rolling out of prevention of mother to child transmission of HIV (PMTCT) while the current study population has examined data over a period of

increasing availability of PMTCT services though the extent of utilisation is not known. To be able to unravel the differences observed, I included the cause of adult death in the exposure variable. It was revealed that the risk of death was highest if a mother died of HIV/AIDS/TB as opposed to other causes when compared with the unexposed children. However, this finding should be taken cautiously as the confidence intervals for the risk estimate for HIV/AIDS/TB deaths and other causes of death overlapped.

The risk of child death was highest in the 6 months following death of a mother but also in the 6 months preceding the death of a mother. Chronic illnesses such as HIV/AIDS slowly and gradually erode the carer role of the mother ultimately resulting in untoward effects on children even before death of mothers occur. This finding is in line with an earlier multi-country study conducted in Tanzania, Uganda and Malawi that showed that risk of child death associated with a mother's death was 3.9 times higher and was centred on the mother's death up to two years.<sup>[17]</sup> Against this background, it appears that most intervention programmes targeting orphans wait for too long to intervene because, as these results show, children of terminally ill mothers have as a high risk of death as those already orphaned.

As expected, the risk of child death decreased with age with the sharpest decline being in the neonatal period. The substantial differences in the risk of death among infants at the various ages have implications in treating under-one year olds as a homogenous age category for both research and intervention purposes. Interventions should pay a lot of attention to the first month of life as the risk of death is highest during this period. As it has been reported in several other studies, male children had a higher risk of death compared with their female counterparts<sup>[309]</sup>. Sex preference is unlikely to explain this result as the predominant form of gender preference in Kenya is the balanced type with roughly equal proportions preferring girls to boys and vice versa<sup>[310]</sup>. As with adults, mortality was highest among the Luo and Luhya ethnic groups. Since for adults, HIV/AIDS is the main driver for the high mortality in these ethnic groups, it is therefore possible that the excess mortality among children is due to paediatric HIV infection but also due to consequences of orphanhood as a result of HIV/AIDS mortality. From these results, it is likely that paediatric HIV infections are a significant contributor to Luo child deaths given that children of mothers who die of HIV/AIDS/TB also have a higher

risk of death. Other possible explanations may include long standing economic differentials by ethnicity that impact on child survival as has been reported elsewhere <sup>[311]</sup>. Studies have demonstrated differentials in utilisation of health care services by ethnicity, for example the Luo and Luhya have been reported to have lower immunisation coverage in the slums compared with the Kikuyu <sup>[198]</sup>.

Children from households categorised as being in the middle category in the wealth index had a lower risk of death than the poorest, but it is not clear why those from the wealthiest households had similar mortality experience to those from the poorest households. This might be related to the observation made elsewhere that HIV or risky sexual behaviours are more prevalent among the wealthier members of society, although this is not yet supported by studies in this population <sup>[312, 313]</sup>.

Surprisingly, maternal education was not significantly associated with child survival in this study. Many research reports have identified maternal education as being an important predictor of child survival <sup>[314, 315, 316]</sup>. It is assumed that maternal education mediates child survival through improved knowledge on child care, better income, better housing, access to cleaner water and proper toilet facilities <sup>[317]</sup>. In the slum population, however, there is limited variability in terms of education with a big majority having partial primary education making it hard to discern the beneficial effects of maternal education. In line with other studies, children who were born by teenage mothers had a higher risk of death than those whose mother were aged between 20 and 39 years of age at the time the child was born <sup>[318]</sup>. Teenage mothers are not socially, psychologically and financially prepared to look after a newborn. The higher risk of developing obstetric complications among teenagers directly translates into poor chances of survival for their newly born babies.

### **Data quality issues/limitations**

While the prospective nature of the demographic surveillance system allows for recording of events and dates of their occurrence accurately, it is not devoid of limitations which might compromise the accuracy and validity of the estimates obtained from the data. Attrition from the demographic surveillance area is a key challenge due to the high mobility of residents. Unlike in many rural settings, residents of informal



settlements change places of abode quite often and tracking those moving can be a logistical nightmare. Loss to follow-up can affect estimates from a population especially when the factors under investigation are related to a tendency to migrate. For example, before the advent of the highly active anti-retroviral therapy for HIV/AIDS, many terminally-ill individuals would retreat to their rural homes before death <sup>[105]</sup>. Results here showed that exposed children were more likely to die but also at the same time they were modestly more likely to out-migrate from the surveillance area. This means that the count of deaths is likely to be underestimated but the extent of this cannot be quantified as individuals are not routinely traced outside of the surveillance area. Similarly, some speculation exists that women from certain ethnic backgrounds retreat to their rural homes where they can get family support to give birth. While this is not established, were it to be true, a proportion of births and deaths would be missed especially those that result into an early child death hence a lower death count. It is important to note here that, as opposed to other surveys like the DHS, in the DSS information on children born and those that die are not derived from a maternal birth history, so children born when the mother is away may not be counted if the length of absence exceeds 120 days.

Lastly, some key classical predictors of child survival often used in surveys are not routinely collected in the NUHDSS. These include among others parental survivorship of parents not resident in the surveillance area, birth order, birth interval, and survivorship of other siblings <sup>[318]</sup>.

### **Conclusions and recommendations**

Infant and under-five mortality in the slums is high. However, it is comparable to the national estimates and those for Nairobi province over a similar period of time. Mortality rates, particularly neonatal mortality rates, are likely to be underestimated owing to the high risk of migration of children who also have a high risk of death (exposed to maternal death).

Death of a mother adversely affects child survival, particularly, in the 6 months before death and in the 6 months following death. Children born to teenage mothers have a higher risk of death. Therefore it is important that steps are taken to reduce the

incidence of teenage pregnancies which are often mistimed and or unwanted. In recent years, utilisation of modern contraceptive services in Kenya has declined. This has led to several problems including increased unwanted pregnancies with their attendant problems, such as complications of unsafe abortions. This trend needs to be reversed through making contraceptive technology readily available and affordable to young women.

While interventions aimed particularly at child health need to be scaled up, a broad approach including improving the health of mothers is critical in order to be able to reduce the risk of child death. This may involve improving maternity health care services and increased access to HIV/AIDS prevention, care and treatment services.

## **9. Cause-specific child mortality and associated factors**

### **9.1 Introduction**

Globally, a short list of causes of death accounts for most child deaths. Nonetheless, great variation exists between regions and within countries. This variation is a very important consideration in planning, resource allocation and intervention implementation. For example, while malaria contributed 8% of the total burden of under-five deaths in 2008, 94% of the global malaria burden occurred in Africa <sup>[158, 159, 160]</sup>. Similarly, in Kenya malaria endemicity and associated mortality varies greatly. All-cause mortality data for children are relatively more available than adult mortality data. However, cause-specific mortality data for children are very rare and there are variations in estimates depending on methodologies and data sources used <sup>[319, 320, 321]</sup>. This lack of appropriate data is a challenge in elucidating and understanding the dynamics of child health with respect to specific causes at a population level. The most widely available data on child health in developing countries are those from national surveys, mainly covering morbidity and health care utilisation, but not mortality. Surveys are also infrequent and may generally not have disaggregated data for sub-populations such as those living in informal settlements.

The active demographic surveillance in the two informal settlements in Nairobi city provides an alternative source of good data albeit on a localised sub-national population. The DSS collects data on births, movements, deaths and causes of death arrived at using verbal autopsy. While the sensitivity and specificity of the verbal autopsy tool for some childhood illnesses, particularly febrile illnesses such as malaria and pneumonia is low, in the circumstances, these data are the best available at a population level <sup>[77, 267, 322, 323, 324]</sup>. This is because cause-of-death data collected in health care facilities are largely incomplete and may not give a true picture of the spectrum of causes of death among children at population level. Individuals who die in health care facilities may be a select group which may not be representative of the general population. Additionally, not all deaths may be properly certified especially in lower health care facilities where there are no doctors. The opportunity provided by the DSS data to study child survival is unique and opens more opportunities for research.

The HIV/AIDS epidemic affects children in various ways. Parental death may result in a child suffering adverse effects of orphanhood in terms of lost care, but also they may acquire paediatric HIV infections from mothers who are infected. At a population level, if many mothers are infected with HIV, it is likely that a substantial number of children will be infected through mother-to-child transmission of HIV since utilisation of PMTCT is still limited. As a consequence, children born to HIV positive mothers are likely to have higher risk of death themselves. Although recent data shows a decrease in the number of cases of paediatric HIV infection, the total number of infections remains high<sup>[302]</sup>. Several reports have reported on the growing crises of orphaned and vulnerable children unfolding in sub-Saharan Africa as a result of high adult HIV/AIDS mortality<sup>[16, 25, 206]</sup>.

The additional value of having data on specific causes of child death in this research is that it allows for further investigation into the relationship between exposure to an adult death and risk of death from the specific causes. The pattern may vary from one cause of death to another. For example, it is not known whether children exposed to an adult death are likely to have the same risk of dying from malnutrition as, say, pneumonia or diarrhoea. Such differences, if they existed, are likely to have implications for targeted interventions and further research. This chapter therefore explores cause of death, cause-specific mortality rates for children less than 5 years of age and identifies risk factors associated with death from the major causes.

## **9.2 Methods and data sources**

### **Data analysis**

Analysis in this chapter is restricted to children who were less than 5 years of age. The exposure variable was as defined earlier. However to facilitate investigation of the role of cause of death of an adult on risk of child death, exposure was additionally re-defined thus; 1) unexposed-no adult death, 2) adult died of HIV/AIDS/TB, 3) adult died of injuries and 4) adult died of other causes. The categories of adult cause of death were limited by the counts for each cause. Descriptive analyses involved calculation of proportions by causes of death, crude, and age-adjusted cause-specific death rates. Individuals who died of a certain cause were denoted as 1 and the rest 0 to facilitate calculation of survival time and cause-specific survival analyses. In the initial analyses

that precede the imputation of missing cause of death, observations with unknown cause of death were proportionately redistributed to the other known causes stratified on sex.

Multivariable analysis of the risk of death of children from a specific cause by exposure status was carried out using piecewise exponential regression adjusting for clustering of child deaths at household-level. Control variables in the models included children's age, sex, ethnicity, slum of residence and maternal education. Too few instances of multiple adult deaths in a single household occurred to merit a separate category (two or more deaths). Where there was more than one adult death in a household, the earlier death was the one considered. Due to limited number of deaths in categories such as "All other communicable" and "all other non-communicable diseases", multivariable analyses were not run for these causes-of-death categories. Also, unlike in the all-cause analyses in the previous chapter, the cause-specific models were kept as parsimonious as possible due to the limited number of cases.

In total, about 23% of all child deaths did not have a known cause of death. I assessed factors associated with not having a known cause of death to find out whether there were systematic differences which could lead to underestimation or overestimation of the risk of death from certain causes depending on how those factors influenced missingness. Also, this being a substantial fraction of the total deaths, I investigated missingness given available data on the cases that have missing cause of death data and also evaluated changes in risk estimates against those obtained from the observed data. I used multiple imputation techniques using a user written programme (*ice*) implemented in STATA working on the assumption that cause of death data were missing at random [255]. Since the variable (cause of death) being imputed is categorical, multinomial regression was used in the imputation using the following predictors: exposure status, age group, slum of residence, ethnicity, wealth status, calendar years, child co-residence with parents, maternal education, maternal age at child birth and the log of child's survival time. Five imputations were carried out. Piecewise exponential regression was carried out on the imputed datasets that were combined using the *mim* programme implemented in STATA statistical software. Results of the regression estimates from the observed and imputed data files are presented alongside each other for each cause of death for ease of comparison.

## 9.3 Results

### Causes of death (CoD)

Table 32 shows the percentage distribution of the leading causes of death among children less than five years of age with and without the “unknown cause of death” category. About 77% of children who died had a cause of death assigned to them while 23% did not have a cause of death (Table 32). Pneumonia, diarrhoea, measles, malnutrition, HIV/AIDS- together with tuberculosis and conditions grouped together as neonatal, were the leading causes of death among children under-five years of age. The prominent disease entities in the category “other communicable diseases” include meningitis and malaria each accounting for 27% of the total 52 cases in this category. For the “Other non-communicable disease” category, the prominent disease entities include anaemia (50%) and other diseases that would have been classed as neonatal were it not for the death occurring after the neonatal period including prematurity, and jaundice from neonatal period (18%) of the 22 cases in this category.

Table 32: Percentage distribution of cause of death among children under-five years of age

Cause of death (CoD)	With unknown CoD		Without unknown CoD
	Number	%	%
Pneumonia	142	19.4	25.3
Diarrhoea	95	13.0	16.9
Measles	37	5.1	6.6
Malnutrition	35	4.8	6.2
HIV/AIDS/TB	24	3.3	4.3
Injury	22	3.0	3.9
Neonatal conditions*	133	18.2	23.7
Other communicable diseases	52	7.1	9.3
Other non-communicable diseases	22	3.0	3.9
Unknown	170	23.2	
<i>No Consensus on CoD (n=48)</i>			
<i>No Verbal autopsy done (n=122)</i>			
Total	732	100.0	100.0

\*Causes of death due to child birth and or cause of death occurring within the first month of life  
CoD- Cause of death

Out of the 170 deaths with unknown cause of death, 28% of them were cases where a verbal autopsy was done but the physicians reviewing the verbal autopsy questionnaire failed to agree on the probable cause of death. In the remaining 72% of all those with

unknown cause of death, no verbal autopsy interview was conducted at all due to respondents' refusal or prolonged absence from home.

The category of neonatal cause was created to emphasise the uniqueness of neonatal causes, its relation to the quality of obstetric care, its amenability to interventions but also to be able to produce results that are comparable to earlier major publications on cause of death among children. The neonatal causes consist of child birth complications such as asphyxia, trauma, and prematurity but also causes that typically occur in neonates such as congenital abnormalities, sepsis, tetanus and pneumonia. This category was the second most important cause of child death in this population after pneumonia.

Table 33 presents percentage distribution of causes of death among neonates with and without the category of “unknown cause of death”. Among neonates, the leading causes of death include prematurity, pneumonia, septicaemia and asphyxia. About 32% of all neonates do not have cause of death assigned to them. About 40% of these were due to failure by the verbal autopsy reviewers to arrive at an agreeable cause of death. This is a higher percentage compared to 23% among all under-fives deaths, highlighting the challenge of arriving at a cause of death among neonates due to the commonly vague symptoms that manifest in this age group.

**Table 33: Percentage distribution of cause of death among neonates**

Cause of death (CoD)	With “unknown CoD”		Without “unknown CoD”
	Number	Percentage	Percentage
Pneumonia	15	11.3	16.7
Diarrhoea	8	6.0	8.9
Prematurity	19	14.3	21.1
Birth injury/asphyxia	13	9.8	14.4
Neonatal jaundice	2	1.5	2.2
Septicaemia	14	10.5	15.6
Other neonatal causes	19	14.3	21.1
Unknown	43	32.3	
<i>No Consensus on CoD (n=17)</i>			
<i>No Verbal autopsy done (n=26)</i>			
Total	133	100.0	100.0

The results in Table 34 show the percentage distribution of cases with unknown causes of death by various characteristics. Note that in classifying cause of death, all neonates were classified in the category of “neonatal causes” even though some neonates (32%)

did not have a specified cause of death as seen in Table 33. For investigating missingness, those neonates with unspecified causes has been grouped under missing cause of death, hence the percentage of 29.1% in Table 34 instead of the 23% seen in Table 32. The highest percentage of children with unknown cause of death was the unexposed children with about 30% and the least were those who had lost a father. Viwandani slum has the higher proportion (35%) of children with unknown cause of death than Korogocho slum (26%). As for calendar year, there seem to be an increasing trend of proportion of children with unknown cause of death from about 18% in 2004, 21% in 2005, 39% in 2006 and 49% in 2007.

**Table 34: Percentage distribution of cases without known cause of death by socio-demographic characteristics**

Variables	Percentage without Cause of death	Total Number
Death of adult (exposure)		
No adult death	29.8	661
Father died	19.1	21
Mother died	24.1	29
Other adult died	23.8	21
Age group		
<1month	32.3	133
1-2months	25.4	63
3-5months	31.7	101
6-11months	25.4	189
12-23months	35.0	140
24-59months	23.6	106
Sex		
Female	32.0	334
Male	26.6	398
Residence		
Korogocho	25.5	447
Viwandani	34.7	285
Ethnicity		
Kikuyu	34.6	162
Luhya	28.6	119
Luo	23.7	283
Kamba	35.6	101
Cushitic	28.6	28
Other	30.8	39
Wealth tertiles		
Poorest	30.4	227
Second tertile	32.6	132
Wealthiest	26.6	365
Unknown	50.0	8
Years		
2003	20.0	160
2004	17.7	136
2005	20.8	144
2006	39.0	159
2007	48.9	133
Co-residence with parents		
Both parents	30.4	563
Mother-HHH	25.3	75



Mother-Not HHH	24.0	25
Father only	25.8	31
None of parents	23.7	38
Education		
None	28.2	39
Primary	28.7	495
Secondary/higher	29.8	158
Unknown	32.5	40
Maternal age at child birth		
<20yrs	29.7	212
20-29yrs	29.5	393
30-39yrs	25.8	93
40+yrs	38.9	18
Unknown	18.8	16
Total	29.1	732

The results in Table 35 provide an assessment of factors associated with a child having an unknown cause of death. After controlling for other covariates, results show that Viwandani residence was associated with 2.1 times higher odds of not having a known cause of death. Calendar years 2006 and 2007 were associated with a 2.7 and 4.1 times higher odds of not having a known cause of death respectively compared with 2003. For the rest of the covariates in the models, the differences were not significant.

**Table 35: Factors associated with missing cause of death from a logistic regression model**

Variables	Unadjusted Odds ratios	Adjusted Odds ratios
Death of adult (exposure)		
No adult death	1.00	1.00
Father	0.55 (0.18,1.67)	0.65 (0.17,2.44)
Mother	0.75 (0.32,1.78)	1.05 (0.42,2.65)
Other	0.74 (0.27,2.04)	1.21 (0.40,3.70)
Age group		
<1month	1.00	1.00
1-2months	0.71 (0.36,1.40)	0.60 (0.29,1.25)
3-5months	0.97 (0.56,1.69)	0.89 (0.49,1.63)
6-11months	0.71 (0.44,1.16)	0.59 (0.35,1.01)
12-23months	1.13 (0.68,1.86)	1.09 (0.62,1.90)
24-59months	0.65 (0.36,1.15)	0.70 (0.38,1.30)
Sex		
Female	1.00	1.00
Male	0.77 (0.56,1.06)	0.86 (0.61,1.22)
Residence		
Korogocho	1.00	1.00
Viwandani	1.55** (1.12,2.15)	2.11* (1.18,3.78)
Ethnicity		
Kikuyu	1.00	1.00
Luhya	0.76 (0.45,1.26)	0.71 (0.40,1.26)
Luo	0.59* (0.38,0.90)	0.66 (0.41,1.08)
Kamba	1.05 (0.62,1.76)	0.71 (0.39,1.29)
Cushitic	0.76 (0.31,1.83)	0.67 (0.23,1.92)
Other	0.84 (0.40,1.79)	0.80 (0.35,1.83)
Wealth tertiles		
Poorest	1.00	1.00
Second tertile	1.11 (0.70,1.75)	0.86 (0.50,1.48)

Wealthiest	0.83 (0.57,1.20)	1.33 (0.81,2.20)
Unknown	2.29 (0.56,9.42)	3.01 (0.62,14.52)
Years		
2003	1.00	1.00
2004	0.86 (0.48,1.54)	0.90 (0.49,1.66)
2005	1.05 (0.60,1.84)	1.03 (0.58,1.83)
2006	2.56*** (1.55,4.22)	2.65*** (1.56,4.50)
2007	3.82*** (2.28,6.40)	4.09*** (2.35,7.13)
Co-residence with parents		
Both parents	1.00	1.00
Mother-Household head	0.78 (0.45,1.35)	0.78 (0.42,1.44)
Mother-Not household head	0.72 (0.28,1.84)	0.42 (0.14,1.20)
Father only	0.80 (0.35,1.82)	1.15 (0.41,3.22)
None of parents	0.71 (0.33,1.54)	0.79 (0.29,2.14)
Maternal education		
None	1.00	1.00
Primary	1.02 (0.50,2.11)	0.73 (0.31,1.72)
Secondary/higher	1.08 (0.50,2.34)	0.82 (0.33,2.07)
Unknown	1.23 (0.47,3.21)	1.21 (0.36,4.01)
Maternal age at child birth		
<20yrs	1.01(0.70,1.46)	1.24 (0.81,1.88)
20-29yrs	1.00	1.00
30-39yrs	0.83(0.50,1.39)	0.96 (0.55,1.69)
40+yrs	1.52(0.57,4.02)	0.95 (0.33,2.79)
Unknown	0.55(0.15,1.97)	0.51 (0.08,3.32)

Table 36 shows the observed and imputed percentage distribution of causes of death by exposure status. Without taking exposure into account, generally the observed and imputed proportions are quite similar with the exceptions of neonatal causes and injury. For neonatal causes, the imputed estimate is about 5.5 percentage points lower while that for injury the imputed is about 1.8 percentage points higher. The proportions among the unexposed both imputed and observed are very similar to that of the whole group. Among those who lost a father, the imputed proportion for injury is much higher than the observed while that for neonatal causes it is lower. For children who lost a mother, the observed and imputed proportion for pneumonia, HIV/AIDS/TB and neonatal causes were higher than the observed in the whole study population. For children who lost an adult other than their parent, they had a higher proportion of malnutrition than in the general study population. The imputed estimate (19%) in this category of exposure was higher than the imputed for the whole group (6.4%).

Table 36: Percentage distribution of causes of death without and with missing causes imputed

Cause of death	Number	Exposure status									
		Observed, n=562	Imputed	Not exposed		Father died		Mother died		Other adult died	
				Observed; n=506	Imputed	Observed; n=17	Imputed	Observed; n=23	Imputed	Observed; n=16	Imputed
Pneumonia	142	25.3	25.7	25.1	25.9	29.4	28.6	26.1	32.4	25.0	22.9
Diarrhoea	95	16.9	17.2	17.0	17.3	11.8	9.5	13.0	12.4	25.0	19.0
Measles	37	6.6	7.4	6.5	7.4	11.8	9.5	4.3	4.1	6.3	5.7
Malnutrition	35	6.2	6.4	6.3	6.1	0.0	0.0	4.3	4.8	12.5	19.0
HIV/AIDS/TB	24	4.3	4.6	4.0	4.6	5.9	4.8	13.0	15.2	0.0	1.9
Injury	22	3.9	5.7	4.0	5.6	11.8	21.0	0.0	0.0	0.0	3.8
Neonatal causes	133	23.7	18.2	23.5	18.0	11.8	9.5	34.8	27.6	25.0	19.0
Other CD	52	9.3	10.0	9.3	9.7	17.6	15.2	4.3	3.4	6.3	8.6
Other NCD	22	3.9	4.8	4.3	5.4	0.0	1.9	0.0	0.0	0.0	0.0
Total	562	100	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

Table 37 shows crude and age-adjusted sex and cause-specific cause death rates among under-fives from both observed and imputed data. The age adjustment was carried out using an internal standard. The first panel on the left has estimates computed with the category of unknown cause of death redistributed proportionately to the known categories in order to avoid having an inflated denominator and a smaller numerator in computing the cause-specific death rates. A total of about 41,083 person-years were accumulated by all children under-five over the study period. The results show that the leading cause of death was pneumonia with a total of 198 deaths and a corresponding cause-specific death rate of about 423 and 540 deaths due to pneumonia per 100, 000 person-years for girls and boys respectively. Other important causes include neonatal causes, diarrhoea, HIV/AIDS and TB, measles and under-nutrition. The second panel in Table 37 show that generally, the death rates computed when the unknown causes are excluded from analysis are lower than those observed when the unknown cause are proportionately re-distributed to known categories. The third panel shows rates with unknown cause of death imputed. It can be seen that generally results from imputation are more comparable to those in the first panel (unknown cause proportionately redistributed). However there are some subtle differences. Imputed rates for measles, malnutrition, HIV/AIDS/tuberculosis, injury and other non-communicable diseases were higher. The crude and adjusted death rates were comparable with minor differences in the various cause-of-death categories.

Table 37: Crude and age adjusted death rates by gender: Panel (A) - unknown CoD redistributed; (B) excluded and (C) imputed

Causes of death	Unknown CoD proportionately redistributed				Cases with unknown CoD excluded				Imputed	
	Person-years	Deaths	CDR	ASDR (95% CI)	Person-years	Deaths	CDR	ASDR (95% CI)	CDR	ASDR (95% CI)
Pneumonia										
Females	20326	86	423.1	424.5(335.3;513.6)	20259	60	296.2	297.2(222.3;372.0)	423.6	428.4(388.8;469.2)
Males	20757	112	539.6	537.7(438.8;636.6)	20680	82	396.5	394.8(309.9;479.7)	508.8	502.8(459.6;546.0)
Diarrhoea										
Females	20326	59	290.3	290.4(216.6;364.2)	20259	42	207.3	207.5(144.9;270.0)	283.2	285.6(253.2;319.2)
Males	20757	74	356.5	356.1(275.3;436.9)	20680	53	256.3	256.0(187.3;324.7)	324.0	321.6(286.8;355.2)
Measles										
Females	20326	25	123.0	122.8(74.7;170.8)	20259	17	83.9	83.9(44.0;123.7)	153.6	154.8(129.6;178.8)
Males	20757	27	130.1	130.3(81.2;179.3)	20680	20	96.7	96.9(54.4;139.3)	232.8	231.6(202.8;260.4)
Malnutrition										
Females	20326	21	103.3	103.4(59.2;147.6)	20259	12	59.2	59.1(25.7;92.6)	115.2	115.2(94.8;135.6)
Males	20757	28	134.9	135.2(85.2;185.2)	20680	23	111.2	111.5(65.9;157.0)	229.2	229.2(200.4;258.0)
HIV/AIDS/TB										
Females	20326	14	68.9	68.8(32.8;104.9)	20259	8	39.5	39.5(12.1;66.8)	156.0	156.0(132.0;181.2)
Males	20757	20	96.4	96.5(54.2;138.7)	20680	16	77.4	77.5(39.6;115.5)	207.6	207.6(178.8;235.2)
Injury										
Females	20326	11	54.1	54.1(22.2;86.1)	20259	6	29.6	29.6(5.9;53.3)	104.4	104.4(84.0;123.6)
Males	20757	19	91.5	91.7(50.5;132.9)	20680	16	77.4	77.5(39.5;115.4)	218.4	217.2(189.6;246.0)
Neonatal causes										
Females	20326	66	324.7	331.0(258.5;403.6)	20259	66	325.8	332.3(259.6;405.1)	325.2	331.2(295.2;366.0)
Males	20757	67	322.8	316.8(247.6;386.1)	20680	67	324.0	317.8(248.4;387.2)	322.8	316.8(283.2;350.4)
Other CD										
Females	20326	38	187.0	187.1(127.7;246.5)	20259	29	143.1	143.4(91.3;195.5)	205.2	206.4(178.8;235.2)
Males	20757	35	168.6	168.2(112.6;223.8)	20680	23	111.2	110.9(65.7;156.2)	148.8	147.6(123.6;170.4)
Other NCD										
Females	20326	14	68.9	69.2(33.0;105.4)	20259	10	49.4	49.7(18.9;80.4)	90.0	90.0(72.0;109.2)
Males	20757	16	77.1	76.8(39.2;114.4)	20680	12	58.0	57.9(25.1;90.6)	90.0	88.8(70.8;106.8)
Total	41083	732			40939	562				

### **Risk of child death by specific causes from observed and imputed data**

Table 38 gives hazard ratio estimates from piecewise exponential regression models both for observed and imputed data for each cause of death category. Models were built around a few key covariates to avoid model over fitting owing to the limited number of observations per cause of death category. Results per cause of death category are described below and comparisons made. At the bottom of Table 38 are regression estimates with exposure redefined in two ways:

- a) “Not exposed-no adult death”; “adult died of HIV/AIDS/TB” and “adult died from other causes”.
- b) “Not exposed-no adult death”; “mother died” and “other adult relative died”.

### **Pneumonia**

The results show that children exposed to an adult death had about 2.7 times higher risk of death due to pneumonia compared with children not exposed. For the imputed data, the hazard was slightly lower and non-significant. With regard to age, children aged 6-23 months were 66% less likely to die of pneumonia compared with children aged 1-5 months. Children two years and above were over 90% less likely to die of pneumonia than children aged 1-5 months. Note that children aged less than 1 month were all classed into neonatal causes and thus that age category does not apply to other causes. Child’s sex, slum of residence, ethnicity and mother’s education level were not significantly associated with higher risk of child death from pneumonia. Breaking down exposure status further showed that from the observed data, children who had been exposed to a non-HIV/AIDS/TB death in household had nearly 3 times higher risk of death from pneumonia than the non-exposed children. Running the model with exposure classified by how child related to the deceased adults showed that death of a mother was associated with more than 5 times higher risk of death from pneumonia while death of other adults was associated with about two times higher risk than non-exposed children.

### **Diarrhoea**

Children who were exposed to an adult death were 2.1 times more likely to die of diarrhoea than children who were not exposed. From the imputed data, the risk is lower

(HR=1.2) and non-significant. Children aged two years and older were over 90% less likely to die of diarrhoea than children aged 1-5 months. Luo, Luhya and Kamba children were more likely to die of diarrhoea than the Kikuyu. The highest risk of death from diarrhoea was among the Luo children with over 8 times higher risk compared with the Kikuyu. Children's sex, slum of residence, and maternal education were not significantly associated with increased risk of child death from diarrhoea. The estimates from the imputed data show that the risks were lower than that from the observed data and also significant for children aged 6-23 months and residents of Viwandani. From the observed data, results showed that death of an adult due to HIV/AIDS or tuberculosis was associated with about 2.7 times higher risk while death of a mother was associated with about 3.3 times higher risk than non-exposed children.

### HIV/AIDS/tuberculosis

From the observed data, the risk of death from HIV/AIDS/TB among children who had been exposed to an adult death was 4.4 times higher than those who were not exposed. The hazard ratios from the imputed data were lower in magnitude and non-significant. Children aged two years and older had higher risk of death due to HIV/AIDS/tuberculosis but this was not significant. While ethnicity was not significantly associated with increased risk of death from HIV/AIDS/TB among children, the imputed data estimates show that the Luo children had about 2.5 higher risk of death from HIV/AIDS and tuberculosis. The rest of the covariates were non-significant. From the observed data it can be seen that death of an adult due to HIV/AIDS/TB was associated with 5.7 times higher risk of child death than non-exposed children but this was non-significant from the imputed data. From both observed and imputed data, death of a mother was associated with a higher risk of a child dying of HIV/AIDS/TB than non-exposed children.

### Injury

Estimates from observed data showed that boys were about 2.7 times more likely to die of injuries than their female counterparts while from the imputed data set, the estimated risk is about 1.9 times higher for boy than girls. Being exposed to an adult death appears to carry a higher risk of death from injuries as per estimates from observed data, but this was not significant. However from the imputed data the risk estimate was

significant (HR=3, p value <0.05). Furthermore, from the imputed data, Luo children had a higher risk of death from injuries than Kikuyu children. Also children who lived in household with an adult death due to causes other than HIV/AIDs/TB and those who lost an adult other than their mother had higher risk of death due to injuries than non-exposed children.

## Malnutrition

Children who were exposed to an adult death were about 77% more likely to die of malnutrition than children who were not exposed. However, this difference was not statistically significant. From the imputed data, the risk was significantly higher for exposed children (HR=2.6, p value <0.05). The risk of death from malnutrition was about 60% lower in children resident in Viwandani slum as compared with those who were resident in Korogocho slum while from the imputed data, the risk is in the same direction but much lower (HR=0.33, p value <0.01). While estimates from observed data showed that children from the Luo ethnic background had about 3 times higher risk of death from malnutrition compared with those from the Kikuyu ethnic background, estimates from the imputed data showed that the difference was not significant. Estimates from imputed data showed that death of an adult due to HIV/AIDs/TB is associated with a higher risk (HR=3.6, p value <0.05) of child death from malnutrition while death of a mother was associated with an even higher risk (HR=11.2, p value <0.001) of child death.

## Measles

The risk of death from measles was not significantly higher among the exposed children (HR=2.4; P value > 0.05). Children aged 6-23 months had a higher risk of death from measles but this was not significantly different from the other age groups. Children from the Luo ethnic background had more than 3.8 times higher risk of death from measles than their Kikuyu counterparts (HR=3.9, p value<0.01). The rest of the ethnic groups did not have significantly different risk from children from the Kikuyu ethnic group. Similarly from the imputed data, children from the Luo ethnic background had a higher risk of death from measles (HR=2.9, p value <0.05) than their Kikuyu counterparts.



## Neonatal causes

From both observed and imputed data, estimates show that neonates who were exposed to an adult death were more than 2.7 times more likely to die than those who were not exposed to an adult death. Adult death due non-HIV/AIDS/TB causes and death of a mother were significantly associated with a higher risk of death in the neonatal period. Child's sex, slum of residence, ethnic background and maternal education were not significantly associated with risk of death from neonatal causes.

In summary, exposed children generally tended to have higher risk of death from every cause although in some cases, such as injury, malnutrition and measles, the risks were not significantly different. Male children and residents of Korogocho generally had higher risk of death compared with their female and Viwandani counterparts, although not all differences were significant.

Table 38a: Risk of death from specific causes: results from piecewise exponential regression models from observed and imputed data

Variables	Pneumonia		Diarrhoea		HIV/AIDS/tuberculosis	
	Observed data HR(95% CI)	Imputed data HR(95% CI)	Observed data HR(95% CI)	Imputed data HR(95% CI)	Observed data HR(95% CI)	Imputed data HR(95% CI)
Exposure						
Not exposed	1.00	1.00	1.00	1.00	1.00	1.00
Exposed	2.72*** (1.52,4.88)	2.23(0.88,5.66)	2.11* (1.04,4.27)	1.18(0.39,3.53)	4.38* (1.40,13.66)	2.17(0.89,5.30)
Child age group						
1-5moths	1.00	1.00	1.00	1.00	1.00	1.00
6-23months	0.34*** (0.24,0.49)	0.34** (0.19,0.62)	0.85(0.52,1.37)	0.33*** (0.20,0.54)	3.50(0.46,26.87)	1.69(0.68,4.18)
24-59months	0.03*** (0.01,0.06)	0.04*** (0.01,0.12)	0.08*** (0.04,0.16)	0.04*** (0.01,0.09)	1.87(0.24,14.49)	0.52(0.19,1.40)
Gender (child)						
Females	1.00	1.00	1.00	1.00	1.00	1.00
Males	1.36(0.97,1.92)	1.20(0.64,2.25)	1.28(0.85,1.92)	1.09(0.67,1.79)	2.03(0.87,4.76)	1.22(0.74,2.01)
Slum of residence						
Korogocho	1.00	1.00	1.00	1.00	1.00	1.00
Viwandani	0.71(0.49,1.04)	1.01(0.48,2.10)	0.62(0.38,1.02)	0.47* (0.23,0.95)	0.42(0.14,1.24)	0.64(0.31,1.31)
Ethnicity						
Kikuyu	1.00	1.00	1.00	1.00	1.00	1.00
Luhya	0.79(0.45,1.38)	0.60(0.15,2.38)	4.54*** (1.89,10.89)	3.12(0.99,9.80)	0.69(0.18,2.56)	1.04(0.36,2.98)
Luo	1.30(0.84,2.02)	1.75(0.90,3.39)	8.40*** (3.77,18.71)	5.36** (2.13,13.49)	0.92(0.32,2.64)	2.54* (1.18,5.47)
Kamba	0.78(0.46,1.33)	1.14(0.53,2.43)	2.97* (1.15,7.67)	2.16(0.65,7.19)	0.87(0.21,3.52)	0.44(0.12,1.63)
Other	0.59(0.31,1.12)	0.85(0.31,2.31)	2.33(0.84,6.49)	1.60(0.50,5.12)	0.59(0.15,2.26)	1.34(0.48,3.75)
Maternal education						
No education	1.00	1.00	1.00	1.00	1.00	1.00
Primary	1.28(0.67,2.46)	1.82(0.54,6.10)	0.93(0.45,1.89)	1.12(0.38,3.33)	0.64(0.22,1.89)	1.06(0.45,2.51)
Secondary or higher	1.69(0.85,3.35)	1.67(0.46,6.07)	1.16(0.53,2.53)	1.36(0.38,4.90)	0.24(0.04,1.30)	0.90(0.34,2.42)
<b>Model with Exposure broken down according to cause of adult death</b>						
Cause of adult death						
Not exposed	1.00	1.00	1.00	1.00	1.00	1.00
Died of HIV/AIDS/TB	2.41 (0.99,5.85)	3.12(0.83,11.81)	2.70* (1.06,6.88)	1.37 (0.30,6.19)	5.73* (1.15,28.59)	2.45 (0.70,8.56)
Died of other causes	2.91** (1.42,5.97)	1.63 (0.38,7.02)	1.66 (0.61,4.55)	0.99 (0.20,4.98)	3.56(0.85,14.94)	1.96 (0.59,6.53)
<b>Model with Exposure broken down according dead adults' relation to child</b>						
Relationship to dead adult						

Not exposed	1.00	1.00	1.00	1.00	1.00	1.00
Mother died	5.09*** (1.96,13.22)	3.58(0.42,30.51)	3.33* (1.00,11.04)	1.59(0.19,13.05)	17.49*** (4.92,62.14)	4.85* (1.44,16.38)
Other adult died	2.07*(1.05,4.11)	1.64 (0.39,6.94)	1.78 (0.78,4.04)	1.04 (0.28,3.82)	1.32 (0.17,9.96)	1.44 (0.43,4.89)

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$

Table 38b: Risk of death from specific causes: results from piecewise exponential regression models from observed and imputed data

Variables	Injury		Malnutrition		Measles		Neonatal	
	Observed data HR(95% CI)	Imputed data HR(95% CI)	Observed data HR(95% CI)	Imputed data HR(95% CI)	Observed data HR(95% CI)	Imputed data HR(95% CI)	Observed data HR(95% CI)	Imputed data HR(95% CI)
Exposure								
Not exposed	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Exposed	2.41(0.51,11.40)	3.00*(1.19,7.52)	1.77(0.55,5.68)	2.60*(1.04,6.54)	2.39(0.85,6.72)	1.77(0.48,6.56)	2.81*** (1.60,4.93)	2.74*** (1.56,4.83)
Child age group								
1-5moths	1.00	1.00	1.00	1.00	1.00	1.00		
6-23months	2.98(0.38,23.22)	1.15(0.46,2.86)	1.97(0.59,6.66)	1.65(0.66,4.11)	2.15(0.64,7.19)	1.58(0.52,4.79)		
24-59months	1.94(0.25,15.03)	0.45(0.17,1.18)	0.69(0.19,2.46)	0.43(0.16,1.13)	0.69(0.19,2.44)	0.52(0.16,1.74)		
Gender (child)								
Females	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Males	2.67*(1.04,6.88)	1.88*(1.03,3.41)	1.98(0.98,3.99)	1.98*(1.07,3.65)	1.20(0.63,2.29)	1.81(0.99,3.30)	0.96(0.68,1.36)	0.96 (0.68,1.35)
Slum of residence								
Korogocho	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Viwandani	1.20(0.47,3.08)	0.76(0.40,1.42)	0.41*(0.17,1.00)	0.33** (0.15,0.74)	0.53(0.25,1.13)	1.14(0.50,2.60)	0.88(0.59,1.30)	0.88 (0.59,1.30)
Ethnicity								
Kikuyu	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Luhya	1.07(0.25,4.47)	0.96(0.35,2.64)	1.23(0.39,3.91)	0.76(0.31,1.88)	1.47(0.45,4.85)	1.45(0.46,4.62)	1.16(0.66,2.07)	1.17 (0.66,2.07)
Luo	2.43(0.79,7.45)	2.36*(1.15,4.87)	3.02*(1.25,7.29)	1.19(0.59,2.42)	3.85** (1.52,9.76)	2.94*(1.11,7.81)	1.61(1.00,2.60)	1.60 (0.99,2.59)
Kamba	0.83(0.20,3.38)	0.48(0.14,1.71)	0.32(0.04,2.49)	0.56(0.18,1.80)	0.32(0.04,2.60)	0.63(0.19,2.13)	1.14(0.66,1.98)	1.15 (0.66,2.00)
Other	0.72(0.13,4.05)	0.68(0.21,2.23)	0.52(0.10,2.57)	0.56(0.19,1.64)	1.74(0.52,5.77)	1.00(0.27,3.71)	0.62(0.31,1.25)	0.62 (0.31,1.25)
Maternal education								
No education	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
Primary	1.25(0.25,6.19)	1.30(0.46,3.63)	1.08(0.38,3.10)	0.79(0.36,1.70)	1.66(0.46,5.94)	1.28(0.34,4.85)	0.64(0.36,1.13)	0.64 (0.36,1.13)
Secondary or higher	0.90(0.15,5.53)	0.46(0.09,2.28)	0.28(0.05,1.52)	0.20(0.04,1.01)	1.77(0.47,6.68)	0.69(0.15,3.22)	0.60(0.31,1.15)	0.59 (0.31,1.14)

<b>Model with Exposure broken down according to cause of adult death</b>								
Cause of adult death								
Not exposed	1.00	1.00	1.00	1.00	1.00		1.00	1.00
				3.55*				
Died of HIV/AIDS/TB	--	1.16 (0.14,9.33)	1.34(0.18,10.09)	(1.04,12.11)	1.38 (0.20,9.76)	1.34(0.15,11.93)	2.20 (0.78,6.16)	2.12 (0.76,5.95)
Died of other causes	4.05(0.88,18.71)	4.27**(1.63,11.19)	2.10 (0.51,8.65)	1.95 (0.52,7.28)	3.15(0.97,10.27)	2.05 (0.48,8.76)	3.15*** (1.67,5.97)	3.10*** (1.63,5.88)
<b>Model with Exposure broken down according dead adults' relation to child</b>								
Relationship to dead adult								
Not exposed	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
				11.22***			7.65***	7.13***
Mother died	--	3.33(0.43,25.44)	3.03(0.41,22.17)	(3.73,33.75)	2.75(0.41,18.39)	--	(3.65,16.02)	(3.39,14.99)
Other adult died	3.13(0.64,15.25)	2.85*(1.06,7.63)	1.46 (0.36,5.93)	0.60 (0.08,4.31)	2.29 (0.70,7.54)	2.28(0.41,12.77)	1.50 (0.67,3.33)	1.49 (0.67,3.31)

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$

## 9.4 Discussion

Pneumonia, neonatal causes, diarrhoea, measles, malnutrition and HIV/AIDS/TB are the leading causes of child death in the two slums. Malaria was conspicuously missing in the top causes of under-five death in this population. This is a sharp contrast with many studies in sub-Saharan Africa that show that malaria is among the leading causes of child death <sup>[159, 160]</sup>. It is also a departure from what the health management information system morbidity records show. In health care facilities, it is likely malaria is over diagnosed since diagnoses are often based on only presence of a fever. Indeed, to support the finding in this study, a recent study showed no evidence of malaria transmission in Nairobi slums <sup>[127]</sup>. As such, malaria is not expected to be a leading cause of death among children in this population. The neonatal category that accounted for about 18% of all causes included conditions related to child birth and other causes occurring in the first month of life. The leading causes of neonatal death are pneumonia, prematurity, birth asphyxia, septicaemia and diarrhoea and are similar to those published earlier by WHO in which it was estimated that neonatal causes globally accounted for 37% of deaths among children less than 5 years of age in 2000-2003 and 41% in 2008 <sup>[159, 160]</sup>.

In this study, it was not possible to assess the contribution of under-nutrition in cases where it was not identified as the cause of death. This is because, in the NUHDSS, only a single cause of death is recorded. Against this background, it is likely that the contribution of malnutrition and probably HIV/AIDS to child mortality is underestimated given that these are less likely to be picked out as causes of death among children than more revealing diseases such as diarrhoea and pneumonia.

That pneumonia is a major cause of child death in the slums and is higher (26%) than the global (19%) and African region (21%) estimate does not come as a surprise <sup>[159]</sup>. Most of the housing structures in the slums have iron sheeting walls with poor ventilation, and no separate cooking areas. Crowding due to limited space is also common and this creates a perfect recipe for respiratory infections. Related to the risk of diarrhoeal diseases is the poor household and environmental hygiene. In the slums, toilet ownership is low and many households dispose of their human waste on rubbish dump sites or in rivers <sup>[325]</sup>. Most households use water from illegally connected water

pipes from the city mains using plastic pipes with high potential of breaking and contaminating the water. All these factors are likely to increase the risk of diarrheal diseases and hence the high number of deaths due to diarrhoea.

Ascertaining cause of death for children using a verbal autopsy is notoriously difficult owing to some of the symptoms being mild, non-specific and the inability of young children to communicate and explain the symptoms to the carers. Indeed in this study, of the 170 child deaths with no known cause of death, 28% were due to ambiguity in the symptoms leading to the physicians being unable to agree on the probable cause of death. The situation was even worse for neonates with 40% of all deaths without a known cause being due to failure by the physicians to agree of the cause given the information in the verbal autopsy questionnaire.

The main contributors to child mortality in developed and Asian countries are the neonatal causes with over 40% being due to neonatal causes <sup>[160, 326]</sup>. However, in this population, infectious diseases remain the principal cause of child mortality though neonatal causes account for a substantial proportion. The high contribution of asphyxia and septicaemia as causes of death among neonates is a pointer to poor obstetric and newborn care in the slums and probably poor maternity health care seeking behaviour among mothers. An earlier study in the same population showed that none of maternity care facilities in the slums could provide basic emergency obstetric care <sup>[308]</sup>. On the other hand even though 70% of women claimed to have delivered at a health care facility, in total less than 50% delivered in a proper maternity facility with facilities and personnel capable of handling obstetric emergencies and newborn resuscitation <sup>[327]</sup>.

Regression results from both observed and imputed data showed that death of an adult in a household was associated with a significantly higher risk of death from pneumonia, diarrhoea, HIV/AIDS/TB and neonatal causes. Further analysis showed that indeed it is the death of the mother that is most important. This finding is in line with results in the previous chapter that showed that exposure to an adult death was associated with increased risk of all-cause child death (note that the causes outlined above constitute the largest percentage of all causes of child death). The excess risk might be related to loss of care offered by adults, particularly health care seeking, while, for the case of

HIV/AIDS, death of a mother due to HIV/AIDS may indicate that the child was also infected and thus likely to die of HIV/AIDS or other complications. Results also showed that if the mother or father of child died of HIV/AIDS/TB, the child was also more likely to die of same illness. That malnutrition was not significantly associated with an adult death was a bit surprising due to potential loss of nutrition brought about a mother's death. However it is important to note that often malnutrition is not fatal especially for older children. Also fatal malnutrition diagnoses are generally more likely to be missed and more dramatic causes such as pneumonia or diarrhoea are picked out instead, as causes of death. The insignificant results might also be related to inadequate numbers of deaths due to malnutrition.

As expected, the risk of death from pneumonia and diarrhoea decreased with age. On the other hand, the risk of death from HIV/AIDS/TB, injuries, malnutrition and measles seemed to increase between 6 and 24 months of age and fell thereafter but the differences were not significant. In line with findings from other studies, the risk of death from injuries among males was high- more than twice as high as that for females. An explanation might be that boys are prone to getting involved in more risky and daring adventure and recreational activities than their female counterparts hence the increased risk of injury.

Like it has been reported elsewhere, this analysis established that ethnicity was strongly associated with certain causes of death but not all. Given the generally poor environmental hygiene in the slums, one would imagine that all children in these communities are likely to experience the same risk of diarrhoeal diseases but this is not the case. Ethnic background may act through cultural, socio-economic and parenting skills such as feeding, home treatment and health care seeking behaviour to influence differential survival by ethnicity. This was, however, not investigated further in this study for lack of appropriate data. Overall, the Luo children were particularly at an increased risk of death to the extent that they also had higher risk of death from malnutrition and measles. Malnutrition, measles and diarrhoea often occur together or one after the other especially among socio-economically deprived populations. Diarrhoea and malnutrition are mainly associated with environmental hygiene, personal

hygiene and feeding practices while measles may be related to lack of immunisation against measles.

### **Data quality and limitations**

One of the key concerns in longitudinal studies is attrition, particularly if the tendency to migrate is related to the factors under investigation. As I noted earlier, residents of informal settlements are quite mobile, moving from one housing unit to another or one slum settlement to another, or returning to rural areas. The dedicated regular four-monthly status updates aims to minimise this but still many individuals are lost to follow up. In the previous chapter, I investigated differences between those lost to follow-up and those present and this chapter took this investigation further to find out whether those individuals with no cause of death data were comparable with those who had cause of death data.

The results showed that residents of Viwandani were more likely not to have cause of death data. Research from previous studies in the same population and results here have shown that residents of Viwandani are generally more mobile than those of Korogocho. It is therefore likely that missing cause of deaths arise due to out-migration of family members or untraceable internal change of residence before a verbal autopsy is done. Furthermore, results showed that the likelihood of not having a cause of death increased with calendar year. This might be attributed to community fatigue but also more to recent instabilities in the communities related to land evictions by government and hence to out-migration and loss to follow-up. The good news is that the tendency not to have cause of death data was not associated with the key exposure variable (death of an adult) implying that the loss to follow-up was unlikely to be systematic with regard to the exposure of interest.

The multiple imputation exercise for both descriptive and multivariable analyses revealed only subtle differences between the regression estimates derived from the observed and imputed data. The imputed data is akin to a complete dataset with all the cases retained in the multiple imputed data sets during analysis. On the other hand, the observed dataset has variables with missing data and complete case analysis deletes these cases during analysis and thus the results from the two datasets may be different.



Also the multiple imputed datasets produce better estimates of the standard errors of the estimates and this might explain some of the differences in the findings, for example where the estimates are similar but their levels of significance are different, as in the case with pneumonia, injury and malnutrition. To the extent that the missing data are missing at random, but not missing completely at random, the distribution of the predicted values of the outcome variable will be different from that in the observed data hence producing different, and probably less biased, estimates from the imputed data. The level of missingness of the value to be imputed is likely to be very important in this scenario.

Although the data used in this analysis are derived from several years of surveillance, mortality events are relatively rare and, as such, the number of deaths for some causes of death by exposure was too limited to allow for building of more complex models. The wide confidence intervals in some of the estimates require that the results should be interpreted with caution. Furthermore, from the death rate estimates, it became apparent that excluding unknown causes of death from analysis seem to affect the numerator more hence reduces the death rates as judged against rates obtained after imputation and redistribution of cases of unknown cause of death to known categories. This is because most childhood deaths occur in the first month and year of life and hence the contribution of person-time to denominator is small.

Many critics of the verbal autopsy tool point to the tendency for it to have low sensitivity and specificity for certain childhood illnesses, particularly febrile illnesses such as malaria and neonatal causes of death <sup>[267, 324, 328]</sup>. Low sensitivity and low specificity of the verbal autopsy tool for childhood causes of death means that a sizeable proportion of deaths probably end up being misclassified. However, in the absence of proper registration and certification of causes of deaths, these data are the best that are available. The alternative of hospital records is worse due to selection bias as many individuals die outside of health care facilities.

It is also important to note that, while the verbal autopsy tool used here has been validated in other populations, this has not been done in the Nairobi Urban Health and Demographic Surveillance System. This would have gone a long way in giving some

assurance on how the tool performs since the local epidemiology of the leading causes of death varies from place to place and yet it is known that knowledge of local epidemiology of diseases may influence the verbal autopsy reviewers' choices of cause of death.

### **Conclusion and recommendations**

The leading causes of child death in the informal settlements are pneumonia, neonatal causes, diarrhoea, HIV/AIDS/TB, malnutrition, measles and injuries. Death of an adult in household does not influence child mortality from every cause in the same way. Causes such as pneumonia, diarrhoea, HIV/AIDS/TB and neonatal causes seem to disproportionately affect children who have lost an adult in their household, particularly a mother. There were also differences in the risk of death from certain causes by ethnic background, particularly for the Luo. These may be related, in part, to paediatric HIV infections, but also involve other causes, as evidenced by high risk of death of children whose mothers did not die of HIV/AIDS/TB.

Given that most leading causes of child death have cost-effective interventions, there is reason to believe that, if these interventions were extended to this population, many deaths could be averted. The integrated management of childhood illnesses promoted by WHO and the Kenyan government should be rolled out to informal settlements. Treatment of pneumonia only requires a timely antibiotic while diarrhoea requires timely rehydration; these interventions are all affordable even for a developing country like Kenya. Cost-effective interventions, particularly immunisation and the Integrated Management of Childhood Illness (IMCI) packages, need to be delivered to this population as they cover the most important causes of childhood death. There is need to intensify efforts to have every pregnant woman deliver with the assistance of a professional care giver to avoid the huge burden of neonatal deaths, many of which are attributable to poor obstetric care. Newborn care practices may also need to be examined as many neonates are dying of causes that are neither child-birth related nor congenital malformations. Conditions such as sepsis and diarrhoea might be directly related to child-care practices but more research need to be carried out to examine this situation. Interventions aimed at stemming maternal death are likely to have a positive

impact on child survival and need to be implemented adequately, such as the provision of emergency obstetric care services, which are largely missing in this community.

One emerging worry related to child survival is paediatric HIV/AIDS. Many women continue to deliver without knowing their HIV status and thus do not benefit from PMTCT programmes. These paediatric infections need to be averted as a matter of urgency by ensuring that more women know their HIV status and can access PMTCT services. Lastly, further research, probably including qualitative approaches, should be carried out to understand why children from the Luo ethnic group have an increased risk of death from diarrhoea, measles and malnutrition.

## **10. Impact of adult death on child immunisation**

### **10.1 Introduction**

Childhood immunisation is one of the most successful public health interventions ever implemented <sup>[329, 330]</sup>. Globally, the number of fatal and debilitating immunisable infections has gone down following the launching in 1974 and implementation of the expanded programme on immunisation (EPI) in most parts of the world <sup>[330]</sup>. Many deaths from immunisable diseases are averted directly through inducing immunity among immunised children but also through herd immunity. When a substantial number of individuals in a population are immunised (high immunisation coverage) against a given infection, the risk of transmission between contacts is low as these are immune. Because of this, even non-immunised children stand a low risk of getting infected. Unfortunately, coverage in many places is low and thus many children remain unprotected <sup>[159]</sup>. This gap translates into a huge burden of child mortality due to immunisable diseases, particularly in sub-Saharan Africa <sup>[331]</sup>.

Though many research reports have examined the relationship between parental death and child survival, there is a dearth of literature on the impact on a parental death or any adult death on child immunisation and its timeliness. Understanding this relationship is important because immunisation itself is important for child survival <sup>[332, 333, 334]</sup>.

According to the Mosley and Chen model on child survival, lack of preventive interventions or actions by individuals may lead to poor health and death <sup>[162]</sup>. If death of a parent or other caring guardian undermines access to immunisation, then lack of immunisation might be part of the reason why orphaned and vulnerable children experience higher mortality since some of the immunisable diseases such as measles are also the major killers of children in sub-Saharan Africa <sup>[159]</sup>.

World leaders have recommitted themselves in improving child survival and set it as one of the Millennium Development Goals (MDG) <sup>[152]</sup>. Current data show that child survival is generally improving. For example it is estimated that between 1990 and 2008, the under-five mortality rate fell from 100 to 72 deaths per 1000 live births in developing countries. At the same time immunisation coverage for measles, which is

one of the three progress indicators, increased from 70% in 2000 to 81% in 2008 <sup>[296]</sup>. While there has been progress, it is projected that many countries in sub-Saharan Africa will not meet the target of reducing under-five mortality by two-third between 1990 and 2015 <sup>[152]</sup>.

The World Health Organisation has developed guidelines and recommendations on childhood immunisation which have been adopted by most countries. A number of initiatives geared towards development and delivery of vaccines at affordable cost such as the Global Alliance on Vaccines and Immunisations (GAVI), the Global Polio Eradication Initiative, and Measles Initiative, have made commendable progress in ensuring that children all over the world receive vaccines <sup>[181]</sup>. In addition to the many health care system challenges in vaccine acquisition and delivery at both the national level and in health care facilities, many individual and household level factors influence uptake of vaccines <sup>[121, 122]</sup>.

Pockets of sub-populations with low vaccine uptake exist and these may act as sources of infections resulting into epidemic outbreaks and an increase in the risk of death of the wider community. The characteristics of these subpopulations need to be understood to inform adjustment of programmes to reach the hard to reach populations. Generally, the Expanded Programmes on Immunisation (EPI) have had a heavy focus on reaching rural populations with urban areas assumed to have an advantage in accessing and utilising health care. However, recent evidence suggests that pockets of urban population, especially slum dwellers, have poor health indicators including immunisation coverage rates <sup>[12, 13, 38, 198]</sup>. The realisation that urban estimates of immunisation coverage might mask disparities between urban non-slum and slum populations has opened up interest in understanding the unique challenges of delivery and uptake of vaccines in informal settlements with high mobility of residents and poor health care infrastructure <sup>[100, 308]</sup>.

The current literature on determinants of child immunisation status has focussed mainly on rural areas and there is very little evidence on informal urban settlements in sub-Saharan Africa. While migrants might be a select group with attributes that may be related to higher use of health-care services such as education, they encounter social

disruption upon migration. Adapting to the new environment and building social networks might take a long time hence predisposing them to poor health <sup>[335]</sup>. Available evidence shows that higher household wealth status, ethnicity, fewer children in the household, attendance for antenatal care, delivery in a health care facility and higher maternal education are associated with a child being immunised <sup>[198, 199, 200, 336, 337, 338, 339]</sup>. There has been very limited assessment of the impact of adult death on childhood immunisation in the literature. Some studies have suggested a negative impact <sup>[204, 205]</sup>, while others have not <sup>[206]</sup>.

This chapter aims at exploring immunisation coverage and its timeliness and how death of an adult in a household relates to both issues. I hypothesise that children who have lost a parent or other carer are less likely to be immunised than those who have not lost an adult in their household and that they are also more likely to receive vaccinations late. This is based on the premise that, even though immunisation services are offered free of charge at the point of delivery, other hidden costs and barriers exist that a parent or guardian has to overcome to have their children immunised, such as transport costs, the travel and waiting time involved in visiting the clinic, knowledge of usefulness of immunisation and knowledge of where to take a child for immunisation <sup>[193]</sup>.

## **10.2 Methods and data sources**

### **Population**

The analysis in this chapter was restricted to children under the age of 24 months. For computing immunisation coverage and identifying risk factors for incomplete or no immunisation, only children aged 12-23 months were considered. This age category was chosen because it represents the age cohort that is expected to have received all the basic vaccines in the childhood immunisation schedule for Kenya. For examining the timeliness of vaccine doses given, an assessment on children less than 12 months of age was also done in addition to that carried out for children aged 12-23 months.

### **Measurements**

The Kenya immunisation programme adapted the World Health Organisation recommended immunisation schedule thus: Bacillus Calmette-Guérin (BCG), a vaccine

against tuberculosis is recommended to be given at birth or at earliest contact with health facility that can provide it; oral polio vaccine (OPV), is a vaccine against poliomyelitis and it is recommended that a first dose should be given at birth (OPV0), a second dose at 6 weeks (OPV1), third dose at 10 weeks (OPV2) and a fourth dose at 14 weeks (OPV3). However for computing coverage, OPV0 is normally excluded as it is not mandatory for it to be given and many of the children who are born outside of a formal health care facility never receive it. Some reports provide coverage estimates both with and without OPV0 and this approach is used here. The pentavalent vaccine is a combination vaccine comprising of vaccines against diphtheria (D), pertussis (P), tetanus (T), hepatitis B (hepB) and haemophilus influenzae type B (hib) abbreviated as DPT/HepB/Hib. It was introduced in Kenya in November 2001, replacing the DPT combination. A total of three doses are given at 6, 10 and 14 weeks of age. Although this is a pentavalent vaccine, for simplicity, throughout the write-up, it will be referred to as DPT vaccine. The last vaccine in the standard childhood immunisation schedule is the measles vaccine. It is recommended to be given at 9 months of age. A child is considered fully immunised if they receive all the eight vaccines outlined above.

Many children are born outside of health care facilities, and consequently do not get OPV0 because the first contact with a health care facility comes weeks after birth when the children are due for OPV1 but not OPV0. Indeed, during data cleaning, it was discovered that some field workers do mistake OPV1 for OPV0 and this was detected through dates of say DPT1 being the same as that of OPV0 which should not be the case. To be able to correct for this error, all cases that had DPT1-3 and OPV0-2 but no OPV3, the statuses were revised such that OPV0 became OPV1, OPV1 became OPV2 and OPV2 became OPV3. This kind of revision has also been carried out in other surveys such as DHS.

### **Main exposure variable**

The main exposure of interest was whether a child under observation lived in a household that experienced an adult death or not. Children were considered exposed from 1 year prior to the date of adult death onwards. This was to help account for possible effects that could have occurred during the terminal stages of the illness of an adult.

## **Data analysis**

### **Immunisation coverage:**

Descriptive analyses include immunisation coverage for children aged 12-23 months and a breakdown of immunisation coverage by year, maternal education, area of residence, ethnicity, household wealth status and exposure status (death of an adult in same household where child lives). Inverse Kaplan-Meier survival curves are used to show proportions vaccinated for each antigen in the recommended immunisation window period. A child was considered to have failed when they got immunised. In this analysis, I used the recommended time bounds to assess coverage in the recommended window (BCG- birth to 8 weeks; OPV1/DPT1- 4 weeks to 2months; OPV2/DPT2-8 weeks to 4 months; OPV3/DPT3-12 weeks to 6 months and measles-38 weeks to 12 months).

For assessing the relationship between exposure and outcome (immunisation status), logistic regression models were fitted with outcome variable defined as a binary variable thus; fully immunised=1 and partially or not immunised at all=0. Those who had not been immunised at all were too few to warrant a separate category.

### **Immunisation timeliness:**

The analysis on timeliness as an outcome was limited to those children who had immunisation cards and thus dates. Children who did not have immunisation cards were excluded as their immunisation status was through respondent recall. Few vaccines were given earlier than recommended and for analytical purposes these were treated as having been given on time. The time lag between the age at which a given vaccine was due and the actual age at which the vaccine was given was used to measure delay. The analyses took two approaches:

- i) A vaccine was considered to have been given on time if it was given within 30 days of the due date but was considered delayed if it was given 30 or more days from due date. This categorisation was necessary because over 90% of cases with immunisation dates got immunised in the recommended time frame, offering little variability to be able to assess how the main exposure of interest relates to delay is immunisation outside the recommended window. Secondly



some countries have adopted slightly different recommended ages for the various vaccines making comparisons for delay across difficult. With a definite time lag used as “delay”, comparison across countries is easier even if vaccines are not given at the same ages. For the vaccine-specific delay, descriptive statistics in form of frequency distribution of delay by various characteristic was carried out. Logistic regression models were used to carry out multivariable analyses with outcome of interest defined as delay (1=vaccine given 30 or more days from due date and 0=no delay- vaccine given on time or within 30 days from due date). Some other studies have also used a similar classification of delay <sup>[186]</sup>. The key exposure variable was presence of an adult death in household controlling for household and child’s socio-demographic characteristics. Choice of covariates included in the model depended on either a significant association at univariate analysis at 10% level of significance or being a known risk factor for childhood immunisation status from the literature.

- ii) The second part of the analysis on delay was the cumulative delay (summation of delay for all the vaccines). A similar approach has been used before <sup>[187]</sup>. In this study, cumulative delay was categorised into three i.e. no delay (vaccine given with 30 days from due date); 1-2 months delay and 3 or more months delay. Partial ordered logistic regression models were fitted to assess relationship between cumulative delay and exposure controlling for other covariates. Partial ordered logistic regression was chosen over ordered logistic regression because it is less restrictive with regard to the assumption of parallel slopes. The model assesses the assumption for each variable and then relaxes the restriction where the assumption is violated and instead uses the generalised ordered logistic regression modelling on such variables. In this analysis, slum of residence and maternal education did not meet the parallel slope assumption. The modelling was carried out using a user written programme, gologit2, implemented in Stata statistical software <sup>[340]</sup>.

### 10.3 Results

Out of the 13,046 children aged 12-23 months, 1,047 (8%) did not have any immunisation record in the database, (Table 39). The proportions of children with no immunisation data were comparable by exposure status i.e. 8.1% among non-exposed

and 7% among the exposed. A slightly higher proportion of children from Viwandani slum, Kamba ethnicity, those who resided with none of their parents, those born to mothers with unknown age at child birth, no known education or wealth status had no immunisation data though the numbers in the last three categories were generally small.

**Table 39: Percentage distribution of individuals with no immunisation data by exposure and socio-demographic characteristics**

Variables	Number without immunisation data	Percentage (without immunisation data)	Total
Exposure status			
Not exposed	1,013	8.1	12,560
Father died	15	8.6	174
Mother died	5	4.6	108
Other adult died	14	6.9	204
Sex			
Female	519	8.0	6,478
Male	528	8.0	6,568
Slum of residence			
Korogocho	392	6.3	6,179
Viwandani	655	9.5	6,867
Ethnicity			
Kikuyu	265	7.6	3,478
Luhya	183	8.3	2,213
Luo	178	6.6	2,691
Kamba	256	9.7	2,650
Cushites	60	6.8	877
Other	105	9.2	1,137
Wealth tertile			
Poorest	330	8.2	4,037
Middle	385	9.7	3,955
Wealthiest	302	6.1	4,956
Unknown	30	30.6	98
Parental co-residence			
Both parents	789	7.4	10,686
Mother-Household head	99	9.1	1,085
Mother-Not household head	44	11.6	379
Father only	27	7.9	343
None of the parents	88	15.9	553
Maternal education			
None	34	5.1	665
Primary	650	7.6	8,522
Secondary/higher	218	7.0	3,138
Unknown	145	20.1	721
Maternal age at birth			
<20yrs	625	8.1	7,756
20-29	249	7.8	3,205
30-39yrs	119	7.1	1,679
40+yrs	13	7.4	175
Unknown	41	17.8	231
Total	1,047	8.0	13,046

Table 40 shows results from logistic regression models assessing factors associated with not having immunisation data. After controlling for socio-demographic variables, death of an adult in household was found not to be associated with not having immunisation

data. Children resident in Viwandani slum had 31% higher odds of not having immunisation data compared with those of Korogocho slum. On the other hand, children who lived with either mother only or none of the parents were more likely not to have immunisation data than those who lived with both parents. Children whose household wealth status was not known were more likely not to have immunisation data as compared with those from the poorest households (OR 4.5, p value <0.001) although this estimate is suspect due to small numbers in this category. Children born to mothers whose education status was not known had higher odds of not having immunisation data than those with no formal education, while those whose mother's age at their birth was not known had 64% lower odds of not having immunisation data compared with those whose mothers were aged 20-29 years. A child's sex and ethnicity were not associated with not having immunisation data.

**Table 40: Factors associated with not having immunisation data; results from logistic regression models**

Variables	Unadjusted OR (95% CI)	Adjusted for individual variables OR (95% CI)	Adjusted for all variables OR (95% CI)
Exposure status			
Not exposed	1.00	1.00	1.00
Father died	1.08 (0.63,1.83)	1.17 (0.68,2.00)	1.20 (0.65,2.20)
Mother died	0.55 (0.23,1.36)	0.62 (0.25,1.52)	0.61 (0.25,1.52)
Other adult died	0.84 (0.49,1.45)	0.97 (0.56,1.68)	0.72 (0.41,1.27)
Sex			
Female	1.00	1.00	1.00
Male	1.00 (0.88,1.14)	0.99 (0.87,1.13)	1.00 (0.88,1.13)
Slum of residence			
Korogocho	1.00	1.00	1.00
Viwandani	1.56*** (1.37,1.77)	1.24* (1.00,1.53)	1.31* (1.05,1.62)
Ethnicity			
Kikuyu	1.00	1.00	1.00
Luhya	1.09 (0.90,1.33)	1.12 (0.92,1.37)	1.16 (0.95,1.42)
Luo	0.86 (0.71,1.05)	0.97 (0.79,1.19)	1.01 (0.82,1.24)
Kamba	1.30** (1.08,1.55)	1.15 (0.95,1.39)	1.20 (0.99,1.45)
Cushites	0.89 (0.67,1.19)	1.03 (0.76,1.38)	1.04 (0.75,1.43)
Other	1.23 (0.97,1.56)	1.11 (0.87,1.42)	1.19 (0.93,1.53)
Wealth tertile			
Poorest	1.00	1.00	1.00
Middle	1.21* (1.04,1.41)	1.11 (0.94,1.32)	1.14 (0.96,1.35)
Wealthiest	0.73*** (0.62,0.86)	0.85 (0.70,1.04)	0.88 (0.72,1.08)
Unknown	4.96*** (3.18,7.73)	5.01*** (3.21,7.82)	4.47*** (2.83,7.05)
Parental co-residence			
Both parents	1.00		1.00
Mother-Household head	1.26* (1.01,1.57)		1.32* (1.05,1.66)
Mother-Not household head	1.65** (1.19,2.27)		1.50* (1.06,2.11)
Father only	1.07 (0.72,1.60)		1.20 (0.76,1.90)
None of the parents	2.37*** (1.87,3.02)		2.44*** (1.76,3.38)
Maternal education			
None	1.00		1.00

Primary	1.53* (1.08,2.18)	1.34 (0.91,1.98)
Secondary/higher	1.39 (0.96,2.01)	1.10 (0.73,1.66)
Unknown	4.67*** (3.16,6.90)	4.25*** (2.76,6.53)
Maternal age at birth		
<20yrs	0.96 (0.82,1.12)	0.91 (0.77,1.07)
20-29	1.00	1.00
30-39yrs	0.87 (0.71,1.07)	0.93 (0.75,1.14)
40+yrs	0.92 (0.52,1.62)	0.97 (0.54,1.75)
Unknown	2.46*** (1.74,3.48)	0.36*** (0.21,0.60)

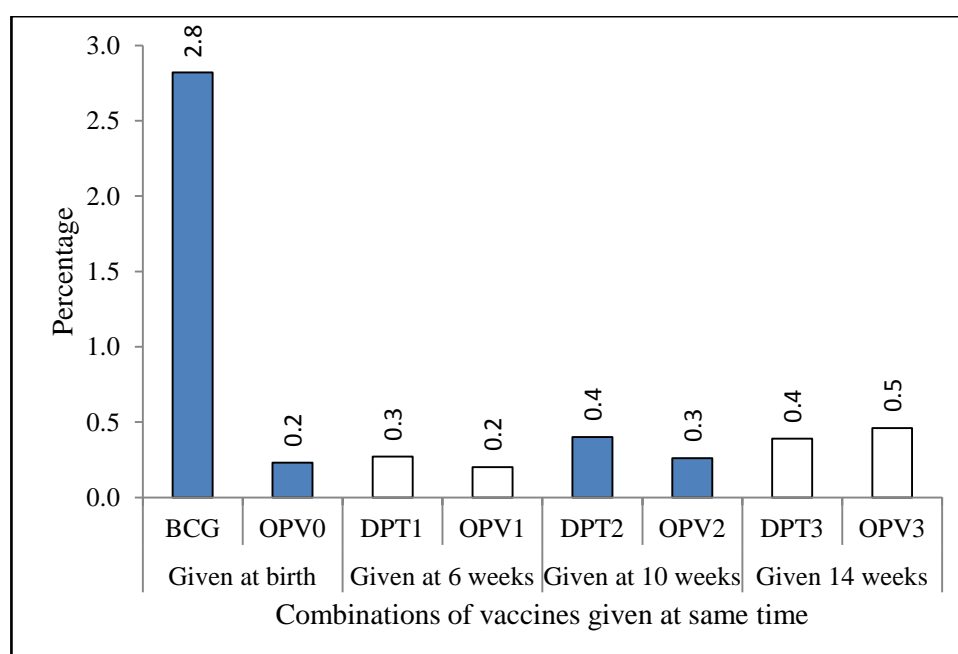
Table 41 shows immunisation coverage for each vaccine and also the full coverage by slum of residence. Results show that for all individual vaccines, coverage was higher in Viwandani slum compared with Korogocho. The proportion of children immunised with a given vaccine decreased with age at which the vaccine is scheduled to be given. For example while 98.4% of all children received BCG (given at birth), 92% received OPV3/DPT3 at 14 weeks and about 80% of children received measles vaccine normally scheduled to be given at 9 months of age. Full immunisation coverage was computed with and without including OPV0. Overall, coverage without OPV0 was 77.7% while that with OPV0 was 73.3%. There were slight differences in coverage by slum of residence. While less than 2% of children never received BCG by the age of 23 months, 20% of children never received measles vaccine by the same age. For all vaccines, higher proportions of children in Korogocho slum provided immunisation status data from immunisation cards as compared with Viwandani. On the contrary, for all vaccines, there were higher proportions of non-immunised children in Korogocho than Viwandani slum.

Table 41: Immunisation (vaccine specific and full) coverage by slum of residence

	Korogocho slum (N=5,786)				Viwandani slum ( N=6,213)				Total (N=11,999)			
	Data source				Data source				Data source			
	Record	Recall	Both	Not immunised	Record	Recall	Both	Not immunised	Record	Recall	Both	Not immunised
BCG	68.9	29.0	97.9	2.1	61.2	37.6	98.8	1.2	64.9	33.5	98.4	1.6
DPT1	67.3	28.7	96.0	4.0	60.0	37.1	97.1	2.9	63.5	33.0	96.6	3.4
DPT2	62.5	31.4	93.9	6.1	57.6	38.1	95.7	4.3	60.0	34.9	94.8	5.2
DPT3	56.1	34.8	90.9	9.1	54.4	39.1	93.5	6.5	55.3	37.0	92.2	7.8
OPV0	57.4	33.0	90.4	9.6	52.6	40.1	92.7	7.4	54.9	36.6	91.6	8.5
OPV1	66.5	29.3	95.8	4.2	59.8	37.2	96.9	3.1	63.0	33.4	96.4	3.6
OPV2	62.0	31.7	93.7	6.3	57.1	38.3	95.4	4.6	59.5	35.1	94.6	5.4
OPV3	55.1	35.5	90.5	9.5	54.1	39.2	93.3	6.7	54.6	37.4	92.0	8.1
Measles	40.4	39.4	79.8	20.2	38.7	41.5	80.3	19.7	39.5	40.5	80.0	20.0
<b>Full coverage</b>												
Without OPV0			76.7				78.6				77.7	
With OPV0			72.0				74.6				73.3	

According to the Kenya national childhood immunisation schedule, some vaccines are scheduled to be given at same age. For example, BCG and OPV0 are supposed to be administered at birth or soon thereafter and therefore it would be expected that BCG and OPV0 coverage should be roughly the same. The same applies to OPV1 and DPT1 scheduled to be given at 6 weeks and so on. Results in Figure 19 assess potential discrepancy in mothers' reporting of vaccines given or actual missed opportunities on the part of service delivery. Results showed that 2.8% of children who received BCG were reported not to have received OPV0 and 0.2% of those who received OPV0 did not receive BCG, (Figure 19).

Figure 19: Proportion of children who received either of two vaccines meant to be given at the same age.



The 2.8% difference observed might be a true reflection of the situation on the ground because many children are not born in health care facilities where vaccination normally takes place and thus miss OPV0 which is recommended to be given within the first four weeks of life. Because of this, children presenting later for immunisation, for example at 6 weeks begin off with OPV1 but not OPV0. On the other hand, although BCG is recommended to be given at birth, children who miss receiving it at birth can receive it at any other time when they make contact with the health care facility hence the discrepancy between the coverage of BCG and OPV0. For DPT1 and OPV1 the differences are much smaller and similarly the difference for DPT2 and OPV2; and

DPT3 and OPV3 are all negligible. This therefore gives us some confidence in the reporting of vaccinations based on mothers' recall.

Table 42 shows immunisation coverage by exposure status and by socio-demographic characteristics. There were very small differences in vaccine specific and full immunisation coverage by exposure status. For all vaccines and full immunisation coverage, children from the Kikuyu ethnic background had slightly better coverage compared with children of other ethnicities particularly the Luo and Cushites, whose coverage was low especially for later vaccines. There were minor differences in coverage by sex, wealth status, and maternal education. Children living with a mother who was the household head and no father had lower immunisation coverage than those born to older mothers (40 or more years at birth).

Table 42: Immunisation coverage by child and maternal characteristics for children 12-23 months old

Characteristics	Vaccines given in the childhood schedule									Coverage (percentage)		Number
	BCG	DPT1	DPT2	DPT3	OPV0	OPV1	OPV2	OPV3	Measles	Coverage without OPV0	Coverage with OPV0	
Exposure status												
Not exposed	98.3	96.4	94.6	92.1	91.5	96.3	94.4	91.8	79.6	77.6	73.3	11,547
Father died	99.4	98.1	94.3	93.1	90.6	98.1	92.5	90.6	81.1	78.6	73.6	159
Mother died	99.0	96.1	92.2	88.4	90.3	96.1	93.2	89.3	80.6	77.7	72.8	103
Other adult died	97.9	96.3	95.3	94.2	92.1	95.8	94.7	93.7	83.2	80.0	76.3	190
Sex												
Female	98.2	96.6	94.7	92.2	91.8	96.5	94.4	91.8	19.9	78.0	73.8	5,959
Male	98.3	96.3	94.5	92.0	91.1	96.1	94.4	91.7	20.1	77.4	72.9	6,040
Ethnicity												
Kikuyu	98.8	97.5	96.2	94.4	94.9	97.3	96.1	94.2	83.9	82.1	79.5	3,213
Luhya	97.9	96.4	94.4	91.0	88.4	96.3	94.2	90.8	78.3	76.4	70.4	2,030
Luo	97.3	94.8	91.7	87.8	87.9	94.6	91.3	87.0	74.2	71.6	66.2	2,513
Kamba	98.8	96.7	95.7	93.8	92.7	96.6	95.5	93.7	80.1	78.3	74.1	2,394
Cushites	98.3	96.3	94.1	91.6	90.9	96.1	93.9	91.3	79.1	76.5	71.4	817
Other	98.4	96.5	95.2	94.0	92.7	96.4	94.9	93.6	82.5	80.8	77.0	1,032
Wealth tertile												100
Poorest	98.4	96.4	95.0	93.0	93.2	96.3	94.7	92.8	80.4	78.5	75.2	3,707
Middle	98.9	97.3	95.9	93.5	92.1	97.2	95.6	93.2	80.8	79.2	74.7	3,570
Wealthiest	97.7	95.9	93.5	90.4	89.6	95.7	93.3	90.0	78.5	75.9	70.8	4,654
Unknown	92.7	88.2	86.8	83.8	88.2	86.8	86.8	80.9	75.0	75.0	70.6	68
Parental co-residence												100
Both parents	98.4	96.6	94.7	92.1	91.6	96.4	94.5	91.8	79.6	77.6	73.2	9,897
Mother-HHH	98.3	96.3	94.1	91.8	90.8	96.2	94.1	91.9	79.2	76.9	72.7	986
Mother-Not HHH	97.6	94.0	92.8	89.3	92.8	94.0	92.2	88.7	75.8	73.7	71.3	335
Father only	98.4	98.1	96.2	94.9	90.8	98.4	95.6	93.4	84.5	81.7	76.9	316
None of the parents	96.8	94.8	94.0	92.9	89.9	95.1	93.8	91.6	82.6	80.7	77.0	465
Maternal education												



None	97.2	95.6	93.8	90.2	89.7	95.1	93.5	90.5	79.9	76.2	70.7	631
Primary	98.3	96.4	94.4	91.8	91.2	96.2	94.1	91.4	79.4	77.5	73.0	7,872
Secondary/higher	98.9	97.2	95.8	93.4	93.1	97.1	95.5	93.2	80.7	78.8	75.0	2,920
Unknown	95.8	94.3	93.2	91.0	88.7	94.4	93.2	90.5	78.3	76.6	72.2	576
Maternal age at birth												
<20yrs	98.4	96.7	95.2	92.5	91.4	96.6	94.8	92.3	81.1	78.7	74.1	2,956
20-29	98.4	96.6	94.7	92.4	91.8	96.4	94.5	92.1	79.8	77.9	73.6	7,131
30-39yrs	98.0	96.0	93.8	91.0	90.8	95.8	93.9	90.6	77.1	74.9	71.0	1,560
40+yrs	94.4	92.6	88.3	80.9	85.2	91.4	87.7	80.3	72.8	69.1	64.2	162
Unknown	95.8	94.2	93.2	92.1	89.5	94.7	93.7	90.5	83.7	82.1	78.4	190
Total	98.3	96.4	94.6	92.1	91.5	96.3	94.4	91.8	79.7	77.7	73.3	11,999

Table 43: Full immunisation coverage (without OPV0) by calendar year

Variables	2003		2004		2005		2006		2007	
	N=3387	Coverage	N=3824	Coverage	N=3814	Coverage	N=3877	Coverage	N=3791	Coverage
Exposure status										
Not exposed	3,248	86.4	3,652	85.4	3,645	83.9	3,733	78.9	3,675	65.9
Father died	52	88.5	57	89.5	52	84.6	52	75.0	40	67.5
Mother died	36	91.7	43	88.4	33	72.7	26	53.9	22	63.6
Other adult died	51	86.3	72	88.9	84	89.3	66	81.8	54	63.0
Sex										
Female	1,726	86.6	1,864	85.7	1,870	84.8	1,920	79.7	1,890	65.5
Male	1,661	86.3	1,960	85.4	1,944	83.2	1,957	77.8	1,901	66.3
Slum of residence										
Korogocho	1,600	82.9	1,882	83.3	1,924	83.0	1,950	80.2	1,804	67.1
Viwandani	1,787	89.6	1,942	87.7	1,890	84.9	1,927	77.4	1,987	64.8
Ethnicity										
Kikuyu	911	90.1	1,039	90.6	1,040	87.8	1,035	83.1	1,050	71.8
Luhya	573	82.2	640	82.0	619	81.6	646	78.8	654	68.0
Luo	729	80.0	845	78.7	823	77.8	805	73.8	728	59.1
Kamba	649	89.4	704	87.4	719	86.8	783	78.0	778	62.5
Cushites	233	88.0	271	84.9	282	82.6	287	79.4	269	62.8
Other	292	91.8	325	90.8	331	86.7	321	78.5	312	68.6
Wealth tertile										
Poorest	1,106	86.4	1,190	86.1	1,133	84.7	1,150	77.2	1,136	67.3
Middle	1,010	91.1	1,123	88.3	1,113	86.3	1,143	79.3	1,157	65.0
Wealthiest	1,246	82.8	1,488	83.3	1,555	81.8	1,570	79.6	1,478	65.4
Unknown	25	80.0	23	73.9	13	69.2	14	71.4	20	75.0
Parental co-residence										
Both parents	2,801	87.4	3,143	85.8	3,122	83.7	3,214	78.5	3,184	65.8
Mother-HHH	259	81.1	288	85.4	325	84.6	333	76.9	305	63.3
Mother-Not HHH	80	81.3	108	75.9	111	78.4	107	81.3	114	65.8
Father only	102	89.2	117	90.6	106	85.9	94	79.8	77	72.7
None of the parents	145	78.6	168	83.9	150	90.7	129	87.6	111	70.3
Maternal education										
None	254	84.3	258	81.8	239	79.9	189	77.3	128	62.5
Primary	2,172	86.0	2,497	85.6	2,481	83.9	2,533	78.9	2,500	65.6

Secondary/higher	831	89.3	913	87.5	908	84.9	934	77.1	944	66.5
Unknown	130	80.0	156	79.5	186	85.0	221	86.0	219	68.5
Maternal age at birth										
<20yrs	947	84.7	1,009	85.2	957	81.8	898	78.7	775	69.4
20-29	1,951	88.0	2,258	86.2	2,251	84.8	2,291	78.8	2,309	64.9
30-39yrs	388	84.8	446	84.5	492	84.2	565	78.2	595	63.9
40+yrs	36	83.3	42	78.6	51	78.4	62	67.7	67	61.2
Unknown	65	78.5	69	79.7	63	88.9	61	93.4	45	91.1
<b>Total</b>		<b>86.5</b>		<b>85.5</b>		<b>84.0</b>		<b>78.8</b>		<b>65.9</b>

Table 43 shows results of full immunisation coverage (computed without OPV0) by calendar year and other socio-demographic characteristics. Overall, full immunisation coverage seems to have reduced over the years with the highest coverage recorded in 2003 at 86.5% and only 65.9% in 2007. With the exception of 2006 and 2007, higher proportions of children in Viwandani were fully vaccinated compared with Korogocho slum. Children from the Kikuyu ethnic background had consistently better immunisation coverage over the years compared with all other identifiable ethnic groups with the Luo having consistently the lowest coverage over the period. Children born to older mothers (40 years and above), had consistently lower immunisation coverage over the years.

Table 44 shows factors associated with full immunisation (without OPV0) status from logistic regression models. After controlling for other covariates, death of an adult in same household where a child lived did not significantly affect the odds of being fully vaccinated. Children from other identifiable ethnic groups were less likely to be fully vaccinated than the Kikuyu counterparts with the Luo particularly being less likely to be fully vaccinated (aOR= 0.54, p value <0.001). Children from households where they did not live with father but mother who was not the household head were less likely to be fully vaccinated and those who lived with only father were more likely to be fully vaccinated compared with children who lived with both parents. The odds of a child being fully vaccinated decreased with the mother's age with those born to mothers aged 40 and above having particularly low odds (38%) of being fully vaccinated compared with those born to mothers aged 20-29 years (OR=0.62, p value<0.01). A child's sex, slum of residence, wealth status of household and mother's education level were not significantly associated with children's full immunisation status.

**Table 44: Factors associated with full immunisation status (without OPV0)**

Variables	Unadjusted OR (95% CI)	Adjusting for child variables OR (95% CI)	Adjusting all variables OR (95% CI)
Exposure status			
Not exposed	1.00	1.00	1.00
Father died	1.06 (0.72,1.55)	1.14 (0.78,1.68)	0.95 (0.62,1.45)
Mother died	1.00 (0.63,1.60)	1.06 (0.66,1.69)	1.07 (0.67,1.71)
Other adult died	1.15 (0.81,1.65)	1.19 (0.83,1.70)	1.16 (0.80,1.67)
Sex			
Female	1.00	1.00	1.00
Male	0.97 (0.89,1.05)	0.97 (0.89,1.05)	0.97 (0.89,1.05)
Slum of residence			
Korogocho	1.00	1.00	1.00
Viwandani	1.11* (1.02,1.21)	0.90 (0.78,1.04)	0.87 (0.75,1.01)
Ethnicity			

Kikuyu	1.00	1.00	1.00
Luhya	0.71*** (0.62,0.81)	0.72*** (0.63,0.82)	0.71*** (0.62,0.82)
Luo	0.55*** (0.49,0.62)	0.56*** (0.49,0.64)	0.54*** (0.47,0.61)
Kamba	0.79*** (0.69,0.90)	0.80** (0.69,0.92)	0.79** (0.69,0.91)
Cushites	0.71*** (0.59,0.86)	0.72*** (0.59,0.87)	0.74** (0.60,0.92)
Other	0.92 (0.77,1.10)	0.94 (0.78,1.13)	0.93 (0.77,1.11)
Wealth tertile			
Poorest	1.00	1.00	1.00
Middle	1.05 (0.94,1.17)	1.06 (0.93,1.20)	1.06 (0.94,1.20)
Wealthiest	0.86** (0.78,0.96)	0.90 (0.79,1.03)	0.89 (0.78,1.01)
Unknown	0.82 (0.47,1.43)	0.86 (0.50,1.51)	0.88 (0.50,1.53)
Parental co-residence			
Both parents	1.00		1.00
Mother-HHH	0.96 (0.82,1.12)		0.92 (0.79,1.08)
Mother-Not HHH	0.81 (0.63,1.04)		0.72* (0.56,0.93)
Father only	1.28 (0.96,1.71)		1.38* (1.01,1.91)
None of the parents	1.20 (0.95,1.52)		1.16 (0.86,1.56)
Maternal education			
None	1.00		1.00
Primary	1.07 (0.89,1.30)		1.04 (0.84,1.30)
Secondary/higher	1.16 (0.94,1.42)		1.08 (0.85,1.36)
Unknown	1.02 (0.78,1.33)		0.82 (0.60,1.11)
Maternal age at birth			
<20yrs	1.05 (0.94,1.16)		1.12* (1.01,1.25)
20-29	1.00		1.00
30-39yrs	0.84** (0.74,0.96)		0.83** (0.73,0.95)
40+yrs	0.63** (0.45,0.89)		0.62** (0.44,0.88)
Unknown	1.30 (0.89,1.89)		1.40 (0.83,2.37)

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ ;

Note: Interaction exposure\* slum; exposure\* ethnicity- not significant

## Timeliness of immunisations

As indicated earlier, assessment of timeliness was based on only children who had immunisation cards and therefore the dates on which the respective vaccines were administered. The proportion of children who had information on dates of immunisation has already been shown in Table 41. Table 45 provides an assessment of factors associated with having immunisation card and thus date when a particular vaccine was given. Estimates are derived from logistic regression models for each vaccine. Results show that after controlling for other covariates, there was no significant association between having an immunisation card and the main exposure (death of an adult in household) for all vaccines. Children from Viwandani slum had lower odds of having an immunisation card with dates for the various vaccines as compared with those of Korogocho. Regarding ethnicity, there were differences for the various vaccines. There was no significant association between ethnicity and having an immunisation card showing dates for BCG. The Luo, Luhya and Cushites had lower odds of having a card with dates for OPV0 compared with the Kikuyu. Luo and Cushites had lower odds of having a card with DPT3 dates while for measles; all other identifiable ethnicities had lower odds of having immunisation card with dates for measles as compared

with the Kikuyu. Children who lived in households where the mother was the household head and no father and those who lived with neither of their parents had lower odds of having immunisation dates for all the vaccines as compared with those who lived with both parents. Those who lived with father but no mother had higher odds of having an immunisation cards as compared with those who lived with both parents.

Figure 20 shows the distribution of ages at which children received the respective vaccines vis-à-vis the recommended age bracket. The recommended age bracket is indicated by the dotted vertical lines in each graph. In a small percentage of children, vaccines were given earlier than recommended as can be seen from the graph for DPT2, DPT3 and measles. This proportion seems to increase with later vaccines. The figure shows that overall, majority of the children receive each of the vaccines within the recommended window period. However, a sizeable proportion of children get immunised after the recommended age as seen by the long tails on the right side of the graphs particularly for DPT3 and measles vaccines. This means that such children remain unprotected for longer periods of time and hence at an increased risk of infection.

Table 45: Factors associated with not having immunisation card/date for each vaccine: results from logistic regression models

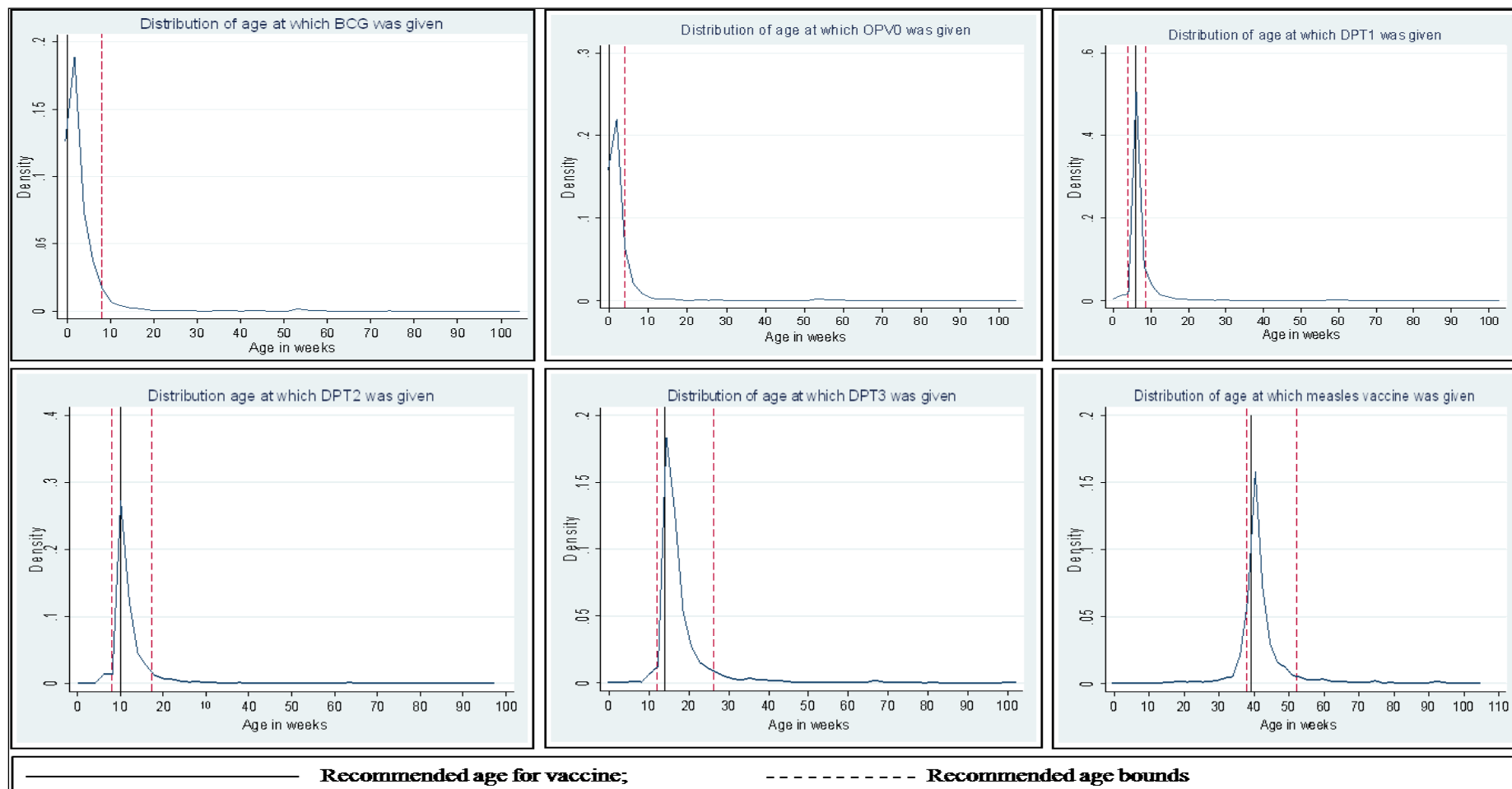
Variables	BCG aOR (95% CI)	OPV0 aOR (95% CI)	DPT1 aOR (95% CI)	DPT2 aOR (95% CI)	DPT3 aOR (95% CI)	Measles aOR (95% CI)
Exposure status						
Not exposed	1.00	1.00	1.00	1.00	1.00	1.00
Father died	1.16(0.79,1.72)	1.18(0.83,1.69)	1.27(0.86,1.87)	1.02(0.71,1.47)	0.99(0.69,1.40)	1.06(0.75,1.50)
Mother died	1.40(0.90,2.16)	1.23(0.83,1.83)	1.29(0.84,1.98)	1.13(0.76,1.69)	1.29(0.86,1.92)	1.12(0.75,1.66)
Other adult died	1.11(0.81,1.52)	1.17(0.87,1.58)	1.31(0.95,1.80)	1.22(0.90,1.66)	1.11(0.82,1.49)	1.03(0.76,1.39)
Sex						
Female	1.00	1.00	1.00	1.00	1.00	1.00
Male	1.01(0.94,1.09)	0.96(0.89,1.03)	1.01(0.94,1.09)	1.01(0.94,1.09)	1.02(0.95,1.10)	1.02(0.95,1.10)
Slum of residence						
Korogocho	1.00	1.00	1.00	1.00	1.00	1.00
Viwandani	0.65*** (0.57,0.74)	0.78*** (0.69,0.88)	0.70*** (0.61,0.79)	0.72*** (0.63,0.81)	0.79*** (0.70,0.89)	0.75*** (0.66,0.85)
Ethnicity						
Kikuyu	1.00	1.00	1.00	1.00	1.00	1.00
Luhya	1.05(0.93,1.18)	0.71*** (0.63,0.79)	1.05(0.93,1.18)	1.00(0.89,1.12)	0.92(0.82,1.03)	0.89* (0.79,0.99)
Luo	1.03(0.92,1.16)	0.76*** (0.68,0.85)	1.03(0.92,1.15)	0.94(0.84,1.05)	0.85*** (0.76,0.94)	0.78*** (0.69,0.87)
Kamba	1.06(0.95,1.19)	0.90(0.81,1.01)	1.03(0.92,1.15)	1.01(0.90,1.13)	0.97(0.86,1.08)	0.86*** (0.76,0.96)
Cushites	1.02(0.85,1.23)	0.81* (0.68,0.96)	1.02(0.85,1.22)	0.92(0.77,1.10)	0.81* (0.68,0.97)	0.71*** (0.59,0.85)
Other	1.12(0.96,1.30)	0.99(0.85,1.15)	1.11(0.96,1.29)	1.09(0.94,1.27)	1.07(0.92,1.24)	1.08(0.93,1.25)
Wealth tertile						
Poorest	1.00	1.00	1.00	1.00	1.00	1.00
Middle	1.17** (1.05,1.30)	0.98(0.89,1.09)	1.15** (1.04,1.28)	1.17** (1.06,1.30)	1.15** (1.03,1.27)	1.15** (1.04,1.28)
Wealthiest	1.04(0.93,1.16)	1.02(0.92,1.14)	1.09(0.97,1.22)	1.02(0.92,1.14)	0.99(0.89,1.10)	0.91(0.81,1.01)
Unknown	0.43*** (0.26,0.71)	0.42*** (0.25,0.70)	0.41*** (0.25,0.67)	0.44*** (0.27,0.72)	0.42*** (0.25,0.71)	0.59(0.34,1.02)
Parental co-residence						
Both parents	1.00	1.00	1.00	1.00	1.00	1.00
Mother-HHH	0.80** (0.70,0.92)	0.82** (0.72,0.94)	0.80** (0.70,0.92)	0.77*** (0.68,0.88)	0.79*** (0.69,0.90)	0.80** (0.69,0.91)
Mother-Not HHH	0.91(0.72,1.15)	0.90(0.72,1.13)	0.88(0.70,1.11)	0.93(0.74,1.16)	0.93(0.74,1.16)	0.77* (0.61,0.98)
Father only	1.39* (1.05,1.85)	1.26(0.97,1.62)	1.35* (1.03,1.79)	1.42** (1.09,1.86)	1.37* (1.06,1.77)	1.17(0.91,1.50)
None of the parents	0.45*** (0.36,0.58)	0.45*** (0.35,0.58)	0.45*** (0.35,0.57)	0.51*** (0.40,0.65)	0.54*** (0.42,0.69)	0.53*** (0.41,0.70)
Maternal education						
None	1.00	1.00	1.00	1.00	1.00	1.00

Primary	0.77*(0.63,0.94)	0.82*(0.68,0.99)	0.86(0.70,1.04)	0.95(0.79,1.15)	1.08(0.90,1.30)	0.95(0.79,1.15)
Secondary/higher	0.84(0.68,1.04)	0.90(0.74,1.10)	0.93(0.75,1.14)	1.05(0.86,1.28)	1.20(0.98,1.46)	1.05(0.86,1.28)
Unknown	0.69*(0.52,0.92)	0.68**(0.52,0.89)	0.76*(0.57,1.00)	0.84(0.64,1.10)	0.96(0.74,1.25)	0.78(0.59,1.03)
Maternal age at birth						
<20yrs	0.94(0.85,1.03)	0.94(0.86,1.02)	0.98(0.90,1.08)	1.00(0.92,1.10)	1.01(0.92,1.10)	1.11*(1.02,1.22)
20-29	1.00	1.00	1.00	1.00	1.00	1.00
30-39yrs	1.03(0.92,1.16)	1.00(0.89,1.12)	1.07(0.95,1.21)	1.01(0.90,1.13)	1.01(0.91,1.13)	0.98(0.88,1.10)
40+yrs	0.80(0.57,1.11)	0.68*(0.49,0.93)	0.74(0.53,1.02)	0.70*(0.51,0.96)	0.67*(0.48,0.92)	0.82(0.58,1.14)
Unknown	1.35(0.88,2.06)	1.53(1.00,2.36)	1.58*(1.03,2.42)	1.39(0.91,2.12)	1.49(0.97,2.28)	1.94**(1.24,3.05)

\*  $p<0.05$ , \*\*  $p<0.01$ , \*\*\*  $p<0.001$



Figure 20: Distribution of ages at which the respective vaccines in the childhood schedule were given



*OPV1, OPV2 and OPV3 not shown because they follow same distribution as DPT1, DPT2 and DPT3 respectively*

Figure 21: Immunisation coverage at various ages among children 12-23 months with immunisation cards

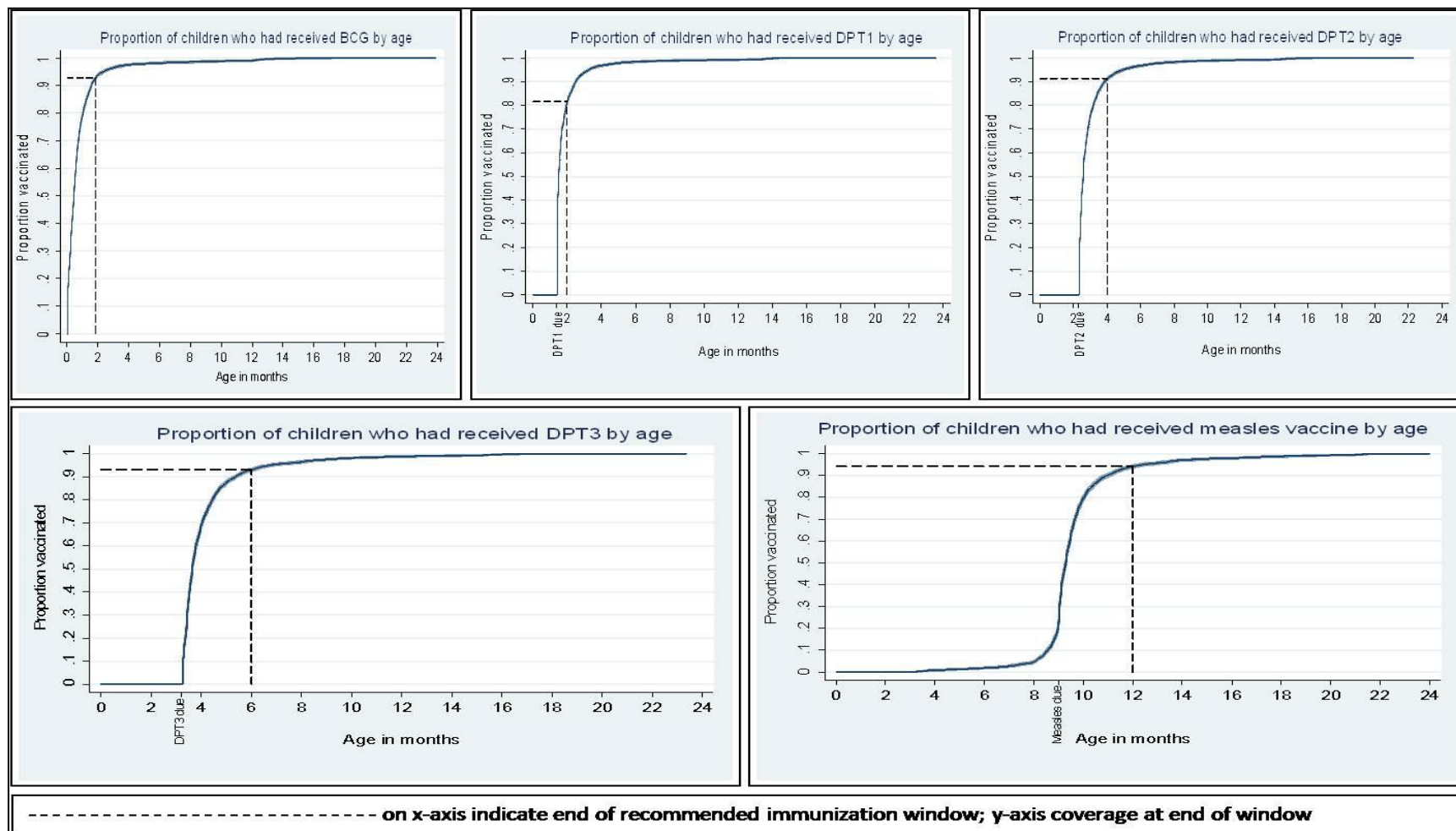


Figure 21 shows the cumulative proportion of children who get vaccinated by end of the recommended immunisation window. Results show that for children who get immunised and have an immunisation record, the majority get vaccinated by the end of the recommended window. About 93% of children receive BCG within 8 weeks of age, 82% receive DPT1 and OPV1 between the recommended age of 4 weeks and 2 months, 91% receive DPT2/OPV2 within the recommended age of 8 weeks and 4 months, 93% receive DPT3/OPV3 within recommended age of 12 weeks and 6 months and 94% of children receive measles within recommended age of 38 weeks and 12 months.

While majority of the children with immunisation records and thus known dates of immunisation get vaccinated within the recommended window period, it is important to assess any delay from the due date as this progressively may result in vaccination taking place outside the recommended window or in eventual default. For this purpose we set delay uniformly and arbitrary at one month from due date as explained earlier. Table 46 shows percentage distribution of children who received the respective vaccines a month or later after due date by exposure status and socio-demographic characteristics. Results show a higher percentage of children from households that had an adult death received vaccinations more than a month after due date as compared with those from households where there was no adult death. A moderately higher proportion of children from Korogocho slum receive their vaccines relatively later than a month from due date compared with those of Viwandani slum. For DPT1 and DPT2 the proportion among Korogocho children is more than twice that for Viwandani children. Children from the Kikuyu ethnic background have the lowest proportion receiving any vaccine later than a month from due date, while the Luhya, Luo and Cushites have the biggest proportions of children receiving vaccines more than a month after due date. Contrary to expectation, a higher proportion of children from the wealthiest households tended to receive vaccines later than a month after due date with those from middle wealth households having the lowest percentage of children receiving vaccines later than a month from due date. A higher proportion of children of mothers with no formal education got vaccinated later than a month after due date than those whose mothers had primary or higher educational. More children born to older mothers received vaccines later than one month from due date than those born to younger mothers.

Table 46: Proportion of children 12-23 months who received respective vaccines more than one month after due date

Variables	BCG		DPT1		DPT2		DPT3		Measles	
	Percentage	Total	Percentage	Total	Percentage	Total	Percentage	Total	Percentage	Total
Exposure status										
Not exposed	21.4	7,459	12.3	7,288	17.8	6,881	25.5	6,345	20.9	4,518
Father died	24.8	117	13.7	117	22.9	105	30.5	95	26.1	69
Mother died	28.0	75	20.8	72	21.5	65	30.2	63	32.6	43
Other adult died	18.4	125	14.7	129	29.2	120	33.0	106	21.9	73
Sex										
Female	20.9	3,852	12.9	3,768	18.1	3,552	24.8	3,263	21.0	2,318
Male	22.0	3,924	12.1	3,838	18.2	3,619	26.5	3,346	21.3	2,385
Slum of residence										
Korogocho	24.8	3,976	16.6	3,882	24.5	3,599	32.8	3,232	24.2	2,310
Viwandani	17.9	3,800	8.2	3,724	11.6	3,572	18.9	3,377	18.2	2,393
Ethnicity										
Kikuyu	10.0	2,053	6.5	2,013	10.3	1,927	17.3	1,822	18.1	1,353
Luhya	32.1	1,326	18.1	1,302	25.5	1,224	34.1	1,114	24.9	798
Luo	30.3	1,675	17.4	1,643	25.4	1,511	34.7	1,345	20.5	927
Kamba	19.1	1,494	8.1	1,451	11.8	1,390	18.6	1,310	19.9	891
Cushites	24.7	566	23.6	547	33.4	494	41.5	424	35.4	288
Other	15.1	662	8.0	650	12.2	625	19.9	594	17.9	446
Wealth tertile										
Poorest	20.5	2,318	12.1	2,257	17.5	2,143	24.9	2,002	20.7	1,438
Middle	17.5	2,242	7.8	2,193	10.9	2,115	18.5	1,997	18.6	1,434
Wealthiest	24.8	3,188	16.0	3,130	23.9	2,888	32.0	2,588	23.4	1,813
Unknown	25.0	28	15.4	26	16.0	25	13.6	22	16.7	18
Parental co-residence										
Both parents	21.6	6,494	12.3	6,352	17.8	5,994	25.3	5,526	21.2	3,951
Mother-HHH	19.7	605	16.0	588	21.8	541	30.7	499	22.2	351
Mother-Not HHH	17.0	212	9.7	206	12.6	199	20.2	183	16.7	120
Father only	21.5	237	12.9	233	19.6	220	27.5	200	22.1	140
None of the parents	24.1	228	10.6	227	20.7	217	28.4	201	19.2	141
Maternal education										
None	25.2	456	26.7	434	34.5	388	47.3	328	31.7	243
Primary	22.9	5,083	12.9	4,978	19.0	4,689	27.1	4,321	21.9	3,057

Secondary/higher	16.1	1,900	8.1	1,860	12.1	1,781	18.3	1,670	16.7	1,202
Unknown	23.7	337	12.6	334	18.5	313	23.5	290	22.4	201
Maternal age at birth										
<20yrs	20.8	4,637	11.3	4,511	16.3	4,271	23.7	3,951	19.9	2,776
20-29	22.3	1,898	14.0	1,875	20.5	1,777	27.4	1,628	21.0	1,201
30-39yrs	21.9	1,042	14.5	1,025	20.5	945	30.7	868	27.4	603
40+yrs	30.0	100	19.4	93	31.3	83	40.9	71	22.2	54
Unknown	21.2	99	9.8	102	17.9	95	23.1	91	17.4	69
<b>Total</b>	<b>21.4</b>	<b>7,776</b>	<b>12.5</b>	<b>7,606</b>	<b>18.1</b>	<b>7,171</b>	<b>25.7</b>	<b>6,609</b>	<b>21.1</b>	<b>4,703</b>

Table 47: Factors associated with immunisation delay (1 or more months from due date) for vaccines given in the childhood schedule: Results from logistic regression models

Variables	Models for various vaccine delays				
	BCG delay aOR (95% CI)	DPT1 delay aOR (95% CI)	DPT2 delay aOR (95% CI)	DPT3 delay aOR (95% CI)	Measles delay aOR (95% CI)
Exposure status					
Not exposed	1.00	1.00	1.00	1.00	1.00
Father died	1.26 (0.77,2.08)	1.12 (0.61,2.07)	1.40 (0.82,2.40)	1.22 (0.73,2.05)	1.44 (0.76,2.72)
Mother died	1.30 (0.77,2.20)	1.62 (0.89,2.93)	1.04 (0.56,1.92)	1.07 (0.61,1.87)	1.72 (0.89,3.31)
Other adult died	0.77 (0.47,1.23)	1.15 (0.68,1.92)	1.69*(1.10,2.59)	1.28 (0.83,1.98)	1.05 (0.59,1.87)
Sex					
Female	1.00	1.00	1.00	1.00	1.00
Male	1.08 (0.96,1.21)	0.93 (0.80,1.06)	1.02 (0.90,1.15)	1.10 (0.98,1.23)	1.01 (0.87,1.16)
Slum of residence					
Korogocho	1.00	1.00	1.00	1.00	1.00
Viwandani	0.73** (0.60,0.89)	0.60*** (0.47,0.78)	0.54*** (0.44,0.68)	0.59*** (0.48,0.72)	0.69** (0.54,0.88)
Ethnicity					
Kikuyu	1.00	1.00	1.00	1.00	1.00
Luhya	4.29*** (3.56,5.18)	3.39*** (2.68,4.28)	3.22*** (2.63,3.94)	2.68*** (2.24,3.21)	1.54*** (1.24,1.91)
Luo	3.73*** (3.10,4.47)	2.83*** (2.25,3.54)	2.71*** (2.23,3.29)	2.39*** (2.01,2.84)	1.11 (0.89,1.38)
Kamba	2.41*** (1.97,2.95)	1.68*** (1.28,2.21)	1.65*** (1.30,2.08)	1.45*** (1.19,1.76)	1.32* (1.05,1.66)
Cushites	2.58*** (1.97,3.37)	3.15*** (2.32,4.26)	3.36*** (2.56,4.40)	2.46*** (1.90,3.20)	2.14*** (1.56,2.93)
Other	1.91*** (1.46,2.49)	1.72** (1.21,2.44)	1.79*** (1.33,2.40)	1.70*** (1.32,2.18)	1.22 (0.91,1.64)
Wealth tertile					
Poorest	1.00	1.00	1.00	1.00	1.00
Middle	1.00 (0.84,1.19)	0.85 (0.67,1.09)	0.83 (0.67,1.03)	0.92 (0.77,1.11)	1.06 (0.85,1.32)
Wealthiest	0.90 (0.77,1.06)	0.88 (0.73,1.07)	0.91 (0.76,1.08)	0.92 (0.78,1.08)	0.95 (0.78,1.16)
Unknown	1.30 (0.54,3.14)	1.33 (0.44,3.99)	0.97 (0.32,2.92)	0.48 (0.14,1.66)	0.86 (0.24,3.00)
Parental co-residence					
Both parents	1.00	1.00	1.00	1.00	1.00
Mother-HHH	1.00 (0.80,1.25)	1.47** (1.15,1.88)	1.42** (1.13,1.78)	1.38** (1.12,1.71)	1.05 (0.80,1.38)
Mother-Not HHH	0.82 (0.56,1.20)	0.82 (0.51,1.34)	0.64* (0.41,1.00)	0.81 (0.55,1.18)	0.78 (0.47,1.28)
Father only	0.86 (0.59,1.24)	0.84 (0.54,1.32)	0.81 (0.55,1.21)	0.87 (0.60,1.25)	0.86 (0.53,1.39)
None of the parents	1.34 (0.89,2.01)	0.81 (0.46,1.42)	1.19 (0.77,1.86)	1.35 (0.89,2.04)	0.99 (0.56,1.74)
Maternal education					

None	1.00		1.00		1.00
Primary	0.98 (0.76,1.27)	0.58*** (0.44,0.76)	0.70** (0.54,0.91)	0.59*** (0.45,0.77)	0.89 (0.64,1.23)
Secondary/higher	0.72* (0.54,0.96)	0.42*** (0.30,0.57)	0.51*** (0.38,0.69)	0.41*** (0.31,0.55)	0.68* (0.47,0.97)
Unknown	1.11 (0.76,1.63)	0.54** (0.34,0.85)	0.61* (0.40,0.93)	0.45*** (0.30,0.68)	0.99 (0.60,1.63)
Maternal age at birth					
<20yrs	1.00 (0.87,1.14)	1.11 (0.94,1.32)	1.14 (0.98,1.32)	1.05 (0.91,1.21)	1.02 (0.86,1.21)
20-29	1.00	1.00	1.00	1.00	1.00
30-39yrs	1.06 (0.89,1.25)	1.15 (0.94,1.42)	1.16 (0.96,1.40)	1.28** (1.08,1.52)	1.42*** (1.16,1.75)
40+yrs	1.51 (0.96,2.38)	1.25 (0.72,2.17)	1.64 (0.99,2.71)	1.61 (0.97,2.67)	0.90 (0.46,1.74)
Unknown	0.72 (0.36,1.46)	1.15 (0.45,2.93)	1.05 (0.48,2.26)	0.92 (0.44,1.88)	0.74 (0.29,1.84)

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$

Table 47 shows results from logistic regression models assessing factors associated with a delay of more than one month from due date for the respective vaccines. Although the odds of receiving vaccines a month or more later than due date were higher among exposed children, this was not significantly different from non-exposed children. Children resident in Viwandani slum were less likely to receive any of the vaccines more than a month late as compared with those of Korogocho. As compared with the Kikuyu ethnic group, children from other ethnic backgrounds had higher odds of receiving vaccines a month after the due date. Wealth status and child's sex did not have significant effect on the timing of vaccination for each of the vaccines. Children from households where the mother was the household head and non-resident father tended to have higher odds of receiving DPT1, DPT2 and DPT3 later than a month from the due date compared with those who lived with both parents. Children born to mothers with primary or higher education had lower odds of receiving DPT1, DPT2 and DPT3 a month or later from due date as compared with those born of mothers with no formal education. Children born to older mothers had higher odds of receiving vaccines later than a month from due date though this was only significant for DPT3 and measles for mothers aged 30-39 years as compared with those born to mothers aged 20-29 years.

Table 48 shows percentage distribution of cumulative delay for all the vaccines by exposure status and socio-demographic characteristics. Children from households that were exposed to an adult death seemed to have slightly higher percentage of children who had a cumulative delay on the eight vaccines of 1-2 or more months. Similarly, a higher proportion of children from Korogocho slum had a cumulative delay of 3 or more months (35%) compared to those of Viwandani (22%). By ethnicity, children of the Luhya, Luo and Cushites ethnicities had higher proportions with cumulative delay of 3 or more months. Children who lived with neither of their parents had the highest proportion that had a cumulative delay of 3 or more months in receiving vaccines. Children of mothers with no formal education and of mothers aged 40 or more years had higher proportions with a cumulative delay of 3 or more months.



Table 48: Cumulative delay in receiving vaccines in the childhood schedule

Variables	Cumulative delay in receiving various vaccines			Total Number
	Less than 1 month	1-2 months	3 or more months	
Exposure status				
Not exposed	35.1	36.8	28.2	7,649
Father died	26.5	40.5	33.1	121
Mother died	26.7	40.0	33.3	75
Other adult died	33.6	29.8	36.6	131
Sex				
Female	35.0	37.2	27.8	3,949
Male	34.7	36.3	29.1	4,027
Slum of residence				
Korogocho	31.0	34.4	34.7	4,077
Viwandani	38.9	39.2	21.9	3,899
Ethnicity				
Kikuyu	43.6	37.3	19.1	2,088
Luhya	26.5	36.5	37.0	1,355
Luo	27.3	36.6	36.1	1,736
Kamba	38.7	38.7	22.6	1,534
Cushites	29.6	29.2	41.2	582
Other	39.8	37.7	22.5	681
Wealth tertile				
Poorest	34.5	37.6	28.0	2,385
Middle	39.1	39.1	21.8	2,290
Wealthiest	32.1	34.5	33.4	3,271
Unknown	36.7	33.3	30.0	30
Parental co-residence				
Both parents	34.8	36.8	28.4	6,658
Mother-HHH	33.8	36.8	29.4	622
Mother-Not HHH	40.9	37.7	21.4	220
Father only	30.9	39.9	29.2	243
None of the parents	36.5	31.3	32.2	233
Maternal education				
None	26.9	28.6	44.5	465
Primary	33.1	37.1	29.8	5,219
Secondary/higher	40.9	38.4	20.7	1,944
Unknown	38.5	32.8	28.7	348
Maternal age at birth				
<20yrs	34.0	35.4	30.6	1,957
20-29	35.8	38.0	26.2	4,743
30-39yrs	31.3	34.7	34.0	1,071
40+yrs	33.0	31.1	35.9	103
Unknown	46.1	29.4	24.5	102
Total	34.8	36.8	28.4	7,976

Table 49 shows results from a partial proportional ordered logistic regression model of how exposure and other socio-demographic characteristics affect cumulative delay in receiving vaccines. After controlling for covariates, results in Table 49 show that although children from exposed households had higher odds of having cumulative delays of a month or more, these were not significantly different from that of the non-exposed children. Children resident in Viwandani had lower odds of having a cumulative delay of a month or more compared to those of Korogocho (OR=0.72, p value<0.001) and they were even less likely to have a cumulative delay of more than 3

months or more as compared with those of Korogocho (OR=0.55, p value <0.001). All ethnicities compared with the Kikuyu had higher odds of having a cumulative delay of 1 or more months. Children from households where the mother was the household head had significantly lower odds of having a cumulative delay of more than one month as compared with those from households with both parents. Children born to mothers aged 30-39 years had 22% higher odds of having a cumulative delay of a month or more as compared with those born to mothers aged 20-29 years. Children from the wealthiest households were less likely to have cumulative delay of one or more months as compared to those from the poorest households (OR=0.84, p value<0.01). Children whose mothers had primary education had 30% lower odds of having a cumulative delay of more than three months as compared with those of mothers with no formal education, however there was no significant difference with a delay of up to 3 months. Children whose mothers had secondary or higher education had 29% lower odds of having a cumulative delay of up to 3 months as compared with those of mothers with no formal education, however the odds were even lower odds of having a delay of 3 or more months (OR=0.5, p value<0.001).

**Table 49: Factors associated with cumulative delay in immunisation:  
Results from partial proportional ordered logistic regression model**

Variables	Less than 1 month Vs 1-2 and 3 + months	Less than 1 and 1-2 months Vs 3 + months
	OR (95% CI)	OR (95% CI)
Exposure status		
Not exposed	1.00	1.00
Father died	1.22 (0.84,1.77)	1.22 (0.84,1.77)
Mother died	1.16 (0.76,1.77)	1.16 (0.76,1.77)
Other adult died	1.14 (0.82,1.59)	1.14 (0.82,1.59)
Sex		
Female	1.00	1.00
Male	1.04 (0.96,1.13)	1.04 (0.96,1.13)
Slum of residence		
Korogocho	1.00	1.00
Viwandani	0.72*** (0.62,0.84)	0.55*** (0.47,0.64)
Ethnicity		
Kikuyu	1.00	1.00
Luhya	2.48*** (2.18,2.83)	2.48*** (2.18,2.83)
Luo	2.09*** (1.84,2.36)	2.09*** (1.84,2.36)
Kamba	1.48*** (1.30,1.69)	1.48*** (1.30,1.69)
Cushites	1.97*** (1.62,2.39)	1.97*** (1.62,2.39)
Other	1.50*** (1.27,1.77)	1.50*** (1.27,1.77)
Wealth tertile		
Poorest	1.00	1.00
Middle	0.99 (0.88,1.12)	0.99 (0.88,1.12)
Wealthiest	0.84** (0.75,0.95)	0.84** (0.75,0.95)
Unknown	1.04 (0.53,2.05)	1.04 (0.53,2.05)
Parental co-residence		

Both parents	1.00		1.00	
Mother-HHH	1.07	(0.91,1.25)	1.07	(0.91,1.25)
Mother-Not HHH	0.74*	(0.57,0.95)	0.74*	(0.57,0.95)
Father only	0.97	(0.75,1.27)	0.97	(0.75,1.27)
None of the parents	1.30	(0.94,1.79)	1.30	(0.94,1.79)
Maternal education				
None	1.00		1.00	
Primary	0.89	(0.71,1.12)	0.70***	(0.56,0.86)
Secondary/higher	0.71**	(0.55,0.90)	0.50***	(0.40,0.64)
Unknown	0.77	(0.57,1.04)	0.77	(0.57,1.04)
Maternal age at birth				
<20yrs	1.03	(0.93,1.14)	1.03	(0.93,1.14)
20-29	1.00		1.00	
30-39yrs	1.22**	(1.07,1.38)	1.22**	(1.07,1.38)
40+yrs	1.05	(0.72,1.53)	1.05	(0.72,1.53)
Unknown	0.64	(0.38,1.09)	0.64	(0.38,1.09)

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$

*Note: Brant test Chi squared=63.49;  $p$  value<0.001(variable slum and maternal education violated the parallel regression line assumption). A significant test statistic provides evidence that the parallel regression line assumption has been violated.*

Table 50 shows results from logistic regression models of the risk of delay in getting vaccinated for the respective vaccines among children of less than 1 year of age.

Controlling for other covariates, it can be seen that the odds of receiving BCG a month or more later among children whose mothers died was about 1.7 times higher than that of non-exposed children. For DPT, the odds of delay were 1.9 times higher for children who lost a mother compared with non-exposed children. For the rest of the vaccines there were no significant associations between exposure and delay. Note that for children aged 12-23, there was no significant association except for DPT2 among children who had lost a non-parent adult in the household. Children less than one year of age from Viwandani slum had lower odds of having a delay in getting vaccinated with any of the vaccines as compared with those from Korogocho. Compared with the Kikuyu, children from other ethnic backgrounds had higher odds of receiving any of the vaccines a month or later. Children who lived with neither of their parents had higher odds of getting vaccinated with BCG a month or more later, but this was not significant for the rest of the vaccines. Children born to mothers with primary or higher education had lower odds of delay as compared with those born mothers with no formal education.

Table 50: Factors associated with immunisation delay for vaccines given in the childhood schedule for children less than 1 year of age: Results from logistic regression models

Variables	BGG N= 12127	DPT1 N= 11464	DPT2 N= 10457	DPT3 N= 9346	Measles N= 6021
	aOR (95% CI)	aOR (95% CI)	aOR (95% CI)	aOR (95% CI)	aOR (95% CI)
Exposure status					
Not exposed	1.00	1.00	1.00	1.00	1.00
Father died	1.21(0.80,1.83)	1.00(0.59,1.70)	1.30(0.82,2.07)	1.04(0.67,1.63)	1.49(0.82,2.72)
Mother died	1.67*(1.08,2.57)	1.91*(1.17,3.14)	1.27(0.75,2.15)	1.10(0.66,1.82)	1.59(0.82,3.06)
Other adult died	0.71(0.47,1.06)	0.89(0.55,1.43)	1.44(0.99,2.10)	1.33(0.92,1.92)	0.92(0.52,1.64)
Sex					
Female	1.00	1.00	1.00	1.00	1.00
Male	1.04(0.95,1.14)	1.02(0.91,1.15)	1.03(0.93,1.15)	1.12*(1.02,1.23)	0.98(0.86,1.13)
Slum of residence					
Korogocho	1.00	1.00	1.00	1.00	1.00
Viwandani	0.81**(0.69,0.95)	0.58*** (0.48,0.72)	0.58*** (0.48,0.69)	0.59*** (0.50,0.70)	0.75*(0.59,0.95)
Ethnicity					
Kikuyu	1.00	1.00	1.00	1.00	1.00
Luhya	3.84*** (3.30,4.47)	3.10*** (2.55,3.75)	3.06*** (2.59,3.61)	2.59*** (2.23,3.02)	1.50*** (1.21,1.84)
Luo	3.14*** (2.71,3.64)	2.50*** (2.07,3.02)	2.35*** (2.00,2.77)	2.15*** (1.86,2.49)	1.10(0.89,1.36)
Kamba	2.32*** (1.97,2.73)	1.64*** (1.31,2.05)	1.51*** (1.24,1.83)	1.41*** (1.19,1.67)	1.29*(1.04,1.62)
Cushites	2.44*** (1.96,3.03)	2.84*** (2.20,3.65)	2.89*** (2.31,3.62)	2.23*** (1.79,2.77)	1.73*** (1.28,2.35)
Other	1.59*** (1.28,1.99)	1.66*** (1.24,2.21)	1.70*** (1.33,2.18)	1.68*** (1.35,2.08)	1.10(0.82,1.48)
Wealth tertile					
Poorest	1.00	1.00	1.00	1.00	1.00
Middle	1.01(0.88,1.16)	0.87(0.71,1.07)	0.83*(0.70,0.99)	0.92(0.79,1.08)	1.14(0.91,1.42)
Wealthiest	1.00(0.88,1.15)	0.88(0.75,1.03)	0.91(0.79,1.05)	0.92(0.80,1.05)	1.05(0.86,1.28)
Unknown	0.98(0.54,1.75)	1.30(0.66,2.55)	0.85(0.42,1.72)	0.65(0.32,1.33)	0.71(0.25,2.05)
Parental co-residence					
Both parents	1.00	1.00	1.00	1.00	1.00
Mother-HHH	0.98(0.82,1.18)	1.52*** (1.24,1.86)	1.31** (1.08,1.58)	1.25*(1.04,1.49)	1.07(0.82,1.39)
Mother-Not HHH	0.98(0.74,1.28)	1.15(0.83,1.61)	0.93(0.69,1.27)	0.89(0.67,1.19)	1.35(0.92,1.98)
Father only	0.91(0.67,1.24)	1.08(0.74,1.56)	0.93(0.66,1.30)	1.04(0.76,1.42)	0.84(0.52,1.35)
None of the parents	1.56** (1.17,2.08)	1.22(0.84,1.78)	1.29(0.93,1.80)	1.28(0.93,1.76)	1.38(0.87,2.18)
Maternal education					
None	1.00	1.00	1.00	1.00	1.00
Primary	0.88(0.72,1.08)	0.59*** (0.47,0.73)	0.66*** (0.53,0.81)	0.62*** (0.50,0.76)	0.85(0.64,1.15)
Secondary/higher	0.65*** (0.52,0.81)	0.45*** (0.35,0.58)	0.51*** (0.40,0.64)	0.45*** (0.36,0.57)	0.69*(0.50,0.96)
Unknown	0.91(0.66,1.24)	0.52*** (0.35,0.76)	0.62** (0.44,0.88)	0.54*** (0.38,0.76)	0.86(0.52,1.41)
Maternal age at birth					
<20yrs	1.06(0.95,1.18)	1.09(0.95,1.25)	1.13(1.00,1.28)	1.13*(1.01,1.27)	0.94(0.79,1.11)
20-29	1.00	1.00	1.00	1.00	1.00
30-39yrs	1.14(0.99,1.30)	1.15(0.96,1.36)	1.19*(1.02,1.39)	1.25** (1.08,1.44)	1.38** (1.12,1.68)
40+yrs	1.32(0.89,1.96)	1.09(0.67,1.79)	1.13(0.70,1.82)	1.43(0.92,2.24)	0.78(0.38,1.62)
Unknown	0.62(0.35,1.08)	1.01(0.49,2.06)	0.98(0.53,1.81)	0.91(0.50,1.64)	0.95(0.43,2.13)

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$ .

Results in Table 51 show that exposure to an adult death was not significantly associated with cumulative delay of one or more months in receiving all the vaccines, Table 51.

Children from Viwandani slum had 25% lower odds of having a cumulative delay of 1-2 months in receiving vaccines as compared with those of Korogocho. Results show that the odds of Viwandani children having a longer delay 3 or more months were even lower (42% lower) to compared to those of Korogocho. Children from the wealthiest

households and those whose mothers had some formal education had lower odds of cumulative delay of 1 or more months in receiving vaccines as compared with those from the poorest and those with no formal education respectively. Children who lived with none of the parents had 33% higher odds of cumulative delay compared with those who lived with both parents while children born to teenage and older mothers had higher odds of delay compared with those born to mothers aged 20-29 years.

**Table 51: Factors associated with cumulative delay in immunisation among children less than 1 year of age: Results from partial proportional ordered logistic regression model.**

Variables	Less than 1 month Vs 1-2 and 3 + months		Less than 1 and 1-2 months Vs 3 + months	
	aOR (95% CI)		aOR (95% CI)	
Exposure status				
Not exposed	1.00		1.00	
Father died	1.20	(0.89,1.63)	1.20	(0.89,1.63)
Mother died	1.31	(0.92,1.86)	1.31	(0.92,1.86)
Other adult died	1.16	(0.88,1.52)	1.16	(0.88,1.52)
Sex				
Female	1.00		1.00	
Male	1.00	(0.94,1.07)	1.00	(0.94,1.07)
Slum of residence				
Korogocho	1.00		1.00	
Viwandani	0.75***	(0.67,0.84)	0.58***	(0.52,0.66)
Ethnicity				
Kikuyu	1.00		1.00	
Luhya	2.26***	(2.04,2.51)	2.26***	(2.04,2.51)
Luo	1.77***	(1.61,1.95)	1.77***	(1.61,1.95)
Kamba	1.36***	(1.22,1.50)	1.36***	(1.22,1.50)
Cushites	1.59***	(1.34,1.88)	2.07***	(1.75,2.45)
Other	1.37***	(1.20,1.57)	1.37***	(1.20,1.57)
Wealth tertile				
Poorest	1.00		1.00	
Middle	0.94	(0.85,1.03)	0.94	(0.85,1.03)
Wealthiest	0.85***	(0.77,0.93)	0.85***	(0.77,0.93)
Unknown	0.79	(0.52,1.20)	0.79	(0.52,1.20)
Parental co-residence				
Both parents	1.00		1.00	
Mother-HHH	1.09	(0.96,1.23)	1.09	(0.96,1.23)
Mother-Not HHH	0.99	(0.82,1.19)	0.99	(0.82,1.19)
Father only	1.09	(0.87,1.35)	1.09	(0.87,1.35)
None of the parents	1.33*	(1.06,1.67)	1.33*	(1.06,1.67)
Maternal education				
None	1.00		1.00	
Primary	0.76***	(0.65,0.89)	0.76***	(0.65,0.89)
Secondary/higher	0.61***	(0.52,0.73)	0.61***	(0.52,0.73)
Unknown	0.68**	(0.53,0.86)	0.68**	(0.53,0.86)
Maternal age at birth				
<20yrs	1.17***	(1.08,1.26)	1.17***	(1.08,1.26)
20-29	1.00		1.00	
30-39yrs	1.14*	(1.03,1.26)	1.14*	(1.03,1.26)
40+yrs	0.99	(0.73,1.33)	0.99	(0.73,1.33)
Unknown	0.75	(0.50,1.13)	0.75	(0.50,1.13)

\* p<0.05, \*\* p<0.01, \*\*\* p<0.001; N=12,669

## 10.4 Discussion

The first objective of this chapter was to explore immunisation coverage and assess how exposure to an adult death affects full immunisation coverage. The second objective was to assess delays in immunisation and factors associated with delay in receipt of the various vaccines in the childhood schedule. Full immunisation coverage stood at 77.7%, quite similar to the national average of 77.4% and slightly higher than that for Nairobi province at 73% over a similar period of time 2003-2007. An earlier smaller study in the same population among children born between 2006 and 2008 estimated a similar coverage at 76.4% <sup>[198]</sup>. Generally, vaccines that come later in the schedule had lower coverage compared with those that come earlier. It appears that the further away from the date of birth the vaccine is scheduled to be given, the higher the chances of defaulting. While coverage for the slums may seem impressive compared with other populations, it remains below optimal coverage. Given the crowding and therefore close personal contact in slums, a higher level of coverage is needed to achieve herd immunity compared with that needed in less congested areas. For example, it is estimated that effective herd immunity for measles can be achieved if immunisation coverage is at least 95% for a country like the United Kingdom while for India a coverage of 99% is needed <sup>[190]</sup>. The required coverage to achieve herd immunity varies by disease depending on the effective reproduction rate,  $R_0$  -number of potential new infections that can result from a single source in a susceptible population <sup>[189]</sup>. For instance, because measles has a high  $R_0$ , it requires a minimal coverage of 95%, while on the other hand it has been established that for haemophilus influenzae type b (hib) disease with a much lower  $R_0$ , a coverage of about 70% is good and has been shown to have been enough to eliminate hib disease in the Gambia <sup>[341]</sup>. In sub-Saharan Africa, low immunisation coverage is commonplace. Given that an additional proportion of children who receive vaccines may not respond, the overall proportion of susceptible children is sizeable and this is recipe for the sustained disease transmission and outbreaks. From a public health perspective, targeting groups with a high risk of transmission, such as slum populations, is potentially beneficial in interrupting transmission and, as a result, a lower coverage would be needed to achieve herd immunity for the wider community in the future <sup>[342]</sup>.

The results in this study showed declining immunisation coverage over the years from 87% in 2003 to about 66% in 2007 and it is not obvious what explains this trend. It could be that immunisation services are deteriorating with fewer children being reached but it could potentially be due to selective participation due to outmigration and refusals to participate in the surveillance over time. However, the latter is unlikely because, as the results show, children with and those without immunisation data were comparable on most of the socio-demographic characteristics.

Based on these results, there was insufficient evidence to conclude that an association exists between death of an adult in a household and an increase in the risk of a child not being fully vaccinated. Given the caring role of most adults, particularly mothers, it might be expected that access to and utilisation of immunisation services would be lower for children exposed to an adult death but the evidence suggests that this is not the case. The likely explanation could be that since coverage is assessed for children aged 12-23 months, there is ample time for catch up on immunisation even for orphaned children. Besides, since orphaned children are more likely to die, they are differentially removed from the risk set used for assessing effect of orphanhood on immunisation. Equally, research carried out elsewhere, although inconclusive, also seems to suggest that orphanhood or death of an adult in household may not be significantly associated with lack of full immunisation <sup>[343]</sup>. One study whose results seem to link lack of vaccination to orphanhood and vulnerability was conducted in Rwanda and used the outcome variable “no vaccination versus at least one vaccination” <sup>[344]</sup>. The problem with grouping say one shot of immunisation together with complete immunisation is that from an immunological and public health point of view these two are very different. Certain vaccines such as DPT and OPV are given as multi-doses at different ages to ensure that the child’s immunity is stimulated enough to produce a strong immune response capable of warding off infection. Therefore grouping children who received one shot of vaccine with those who received three may not be the best measure of immunisation status.

Other important predictors of full immunisation coverage in this population include ethnicity, maternal education and maternal age. This study, like other studies, showed

that the Kikuyu were more likely to be fully immunised compared with other ethnicities [198, 215]. Ethnic differentials in health outcomes in Kenya are common. For example, nationally, the HIV prevalence among individuals aged 15 to 49 among the Luo stand at 20.2%; 4.1% among the Kikuyu and only 0.8% among the Somali [13]. Other indicators such as maternal health care services utilisation also show ethnic divide [327]. The Luo, who also exhibit one of the highest HIV prevalence, child mortality and adult mortality, had the lowest immunisation coverage. Ethnicity is not only an embodiment of cultural practices including those related to child care; there are also noticeable differences in educational attainment and economic empowerment of individuals along ethnic lines. In Kenya, it is known that generally the Kikuyu are economically more empowered than the other ethnicities but it is hard to extrapolate this to the slum population that looks overtly similar in terms of the economic opportunities it offers all ethnic groups. In fact, full immunisation coverage was not associated with the wealth status index. Compared to mothers aged 20-29 years, teenage mothers were more likely to have their babies fully immunised while older mothers, that is those aged 30 years or more, were less likely to have their babies fully immunised. Speculatively, it might be that teenage mothers are more informed about the benefits of immunising their children than older mothers since cost is unlikely to be a barrier.

Assessment of timeliness of vaccines is seldom carried out, yet coverage alone gives an incomplete picture of the state of immunisation in a population. Immunisation coverage may mask disparities in immunisation since children immunised late have a window when they are unprotected and are potentially at risk of getting infected. Unlike coverage, the results here show that delay is not closely related to timing of the vaccination. For example, for BCG which is given at birth, the proportion of children vaccinated a month or later is more than that for DPT1, DPT2 and similar to that of measles given at 9 months. Therefore, it does not follow that the later vaccines in the schedule are necessarily more delayed. This finding is similar to that observed in a multi-country study that showed that the median delay for DPT3 was 6.2 weeks compared to 2.7 weeks for the measles vaccine [180]. One of the key challenges in assessing timeliness of immunisations is the lack of accurate dates when vaccines are given. In some studies this has been as low as less than 50% of the study population and in some cases authors have been forced to impute dates based on available predictors



<sup>[180]</sup>. In this analysis, it was thought that predictors for vaccination dates were not good enough to allow for accurate imputation of dates and hence only individuals with dates were assessed for delay. Because of this, it is possible that children who had immunisation cards and thus used in this part of analysis may have been a select group. For example only about 40% of measles data was from immunisation cards compared with 61% in the KDHS 2008/9 <sup>[13]</sup>.

Loss of an adult in a household was not associated with the risk of delayed receipt of the respective vaccines. Exposed children have a higher risk of delay but this difference was not significant. However, when delay is restricted to children less than 1 year of age, a weak association exists between loss of a mother and delay in receiving BCG and DPT1 in the first year of life but not for the other vaccines. For all children less than 2 years of age, slum of residence, ethnicity, maternal education and age were associated with delay in receiving vaccines. As highlighted in section 3.1, while the two slums are similar in many ways, there are also subtle differences which might translate into the observed differences in immunisation coverage and timeliness. Viwandani slum is the better-off slum with people of slightly higher socio-economic status, which in itself has been associated with higher immunisation coverage and education, and these differences might translate into better health care utilisation <sup>[186]</sup>. Findings showing significant differences in other health indicators/outcomes between the two slums have been reported <sup>[234, 327]</sup>. Children from the wealthiest families had lower chances of a cumulative delay of a month or longer. Similar results associating poverty with delay in getting immunised have been observed in other studies <sup>[186]</sup>. These findings are in line with earlier studies that found delays the receipt of vaccines by children born to mothers with low education, low or advanced maternal age, and in poor and large households in diverse populations <sup>[186, 187, 345]</sup>.

### **Data quality and limitations**

A sizeable proportion of immunisation data was obtained through recall. The extent of recall bias cannot be quantified. First, there is potential for mothers to give socially desirable answers to field workers regarding child care, so it is possible that some mothers might lie about the immunisation status of their children. Secondly, it is

possible that assessment of delay in this study is biased as a substantial proportion of children's immunisation status data was from mothers' recall and therefore excluded from the assessment of delay. This could potentially result in selection bias especially if the children with no immunisation record are likely to be those whose guardians do not pay as much attention to their children's well-being and tend to have children immunised late or opportunistically when they go to a health facility for other medical reasons. If this were the case, results in this study are likely to be biased with higher participation for children with better immunisation histories.

## **Conclusions**

Immunisation coverage in the slum population, although comparable with that of Nairobi province as a whole, still remains suboptimal. Death of an adult in a household adversely affects neither full immunisation status of children nor how soon vaccines are given. However, for children less than one years of age, there is weak link associating death of a mother with delay in receiving BCG and DPT1. Other important correlates of immunisation status and delay include slum of residence, ethnicity, maternal age and education, wealth status and co-residence with parents. These factors should receive consideration in designing of health education messages and also in targeting hard-to-reach people in the community to ensure that the number of unimmunised children is reduced.

## **11. Impact of adult death on children's educational attainment**

### **11.1 Introduction**

Education is a key ingredient in the process of human capital development that is required for economic and social development <sup>[346, 347]</sup>. The Kenyan government has made several attempts at promoting universal primary education since attaining independence in 1963. Indeed, the literacy level in Kenya is among the highest in the African region. The most recent universal primary education policy came into effect in 2003. Its introduction led to a dramatic increase in school enrolment but this has not been sustained over the years, particularly in the informal urban settlements of Nairobi city <sup>[208, 348]</sup>.

Many obstacles need to be overcome to improve children's schooling including provision of adequate physical infrastructure, trained teachers and adequate instruction materials. At the household level, lack of basic necessities such as clothing, housing, nutrition, family stability, and parental guidance may all affect a child's schooling <sup>[212, 349]</sup>. This implies that, even if the educational system is functioning well, some children may still miss out on schooling for reasons outside of the school system. Limited household resources may dictate that not all children go to school or restrict how long they attend school. Expectations of return on investment by the guardians might also inform child schooling decisions. Important too, is how guardians perceive the value of, and how they prioritise, education as a worthwhile venture to spend money on.

This chapter explores the relationship between adult death in a household and children's educational attainment in order to assess whether death of an adult, particularly a parent, erodes the household-level inputs and support that enable child schooling. The impact of adult death on child education has been found to vary from place to place. Some studies have shown negative effects while others found no difference. Earlier research in the slums has shown that children from this population have poorer enrolment rates than children from non-slum urban Nairobi <sup>[350]</sup>. Therefore, given the underlying schooling disadvantage for slum children, it is likely that death of an adult further

negatively impacts on children's schooling. The variations in schooling outcomes noted in various populations emphasise the need for context-specific evidence on the impact of orphanhood to enable the formulation of sound recommendations for policy.

The impact of adult deaths on children's schooling in urban informal settlements in Kenya is yet to be examined. However, given the high adult mortality in the slums, coupled with pre-existing schooling disadvantage, I hypothesise that adult death negatively impacts child education outcomes. This chapter aims at examining the relationship between several measures of educational attainment and death of an adult in the household in two Nairobi city informal settlements controlling for several covariates.

## **11.2 Methods and data sources**

### **Measurements**

#### **Outcome measures:**

No single measure of school participation adequately captures all the different facets of schooling outcomes at an individual level. Bearing this in mind, the following measures of attainment at school have been considered in this analysis:

- i) School enrolment by age 7 (never enrolled compared with enrolled by age 7)  
The analysis here concerns and is limited to children aged 6 and 7 years and their school enrolment status. This was done after preliminary analysis showed that the majority of children enrol in school at one time or another, some of them only for a short duration or at much older ages. Children who were never enrolled in school or enrolled after age of 7 were all grouped together as not enrolled by age 7, while those who enrolled at age 6 or 7 were classified as enrolled by age 7. The variable took on values of 1 if enrolled by age of 7 and 0 if never. Since the outcome variable was binary, logistic regression models were used to assess effect of death of an adult in household on school enrolment by age 7.
- ii) Grade repetition  
Children who for one reason or another were in the same grade in two different calendar years were considered to have repeated the grade in question. The academic year is

coterminous with the calendar year and this makes assessment of progression a lot easier. There were a few instances of multiple repetitions but for analytical purposes, only the last episode was considered. The variable took on values of 1 if a child had ever repeated a grade and 0 if he or she had not. Logistic regression models were used to assess relationship between death of an adult and grade repetition.

iii) Interruption of schooling among those enrolled

Children were considered to have had their schooling interrupted if they were out of school in a particular entire calendar year. The variable took on values of 1 if the child was not in school and 0 if in school for a given academic year. An assessment of the appropriateness of using either fixed effects or random effects using Hausman test was carried out. The test showed that the coefficients were not significantly different and thus random effects models were used to fit the schooling interruption outcome.

iv) Inappropriate grade-for-age

Since children are expected to join grade one at the age of six, they are expected to clear grade eight before they reach age 14. However progression in schooling may not be as expected for a number of reasons. A child may not be in an appropriate grade-for-age because they enrolled in school later than recommended; it might also be due to grade repetition; or dropping out of school for some time and re-joining later. All these may leave a child in an inappropriate grade-for-age. This outcome was evaluated at the last time that the child was observed. The variable took on values of 1 if the child was in an inappropriate grade-for-age at the end of observation and 0 if child was in the appropriate grade-for-age. Few children enrolled earlier than recommended and, for analytical purposes, they were considered to have enrolled at the correct age (6 years).

**Main exposure variable:**

The explanatory variable (exposure status) was defined as a child living in a household that experienced an adult death categorised as: i) not exposed; ii) father died; iii) mother died; and iv) non-parental adult death in the child's household. Exploratory analyses were conducted in which the exposure was defined differently taking into account the cause of adult death with the following categories (non-exposed, adult died of HIV/AIDS; adult died of injuries and adult died of other causes). However, there were

no significant differences by causes of death and therefore this elaboration of the analysis was not pursued further and the results are not presented here.

Furthermore, multiple imputations for missing data were carried out on all outcomes for cases that were lost to follow-up before an event such as school enrolment, grade repetition, dropping out or not being appropriate grade-for-age occurred. This was done because, if the individuals had not been lost, the eventual outcome might have differed from that when they were last observed. The imputation approach used assumes that data are missing at random and that the known information from the cases with missing data can be used to predict the outcomes. The variables used in the analytical models were the same as those used in the imputation. Results from the imputation exercise showed negligible differences in the estimates from those made from the observed data and therefore results from the imputed datasets are not presented.

Proportions of children ever-enrolled, who had ever-repeated a grade, ever-interrupted schooling and had an inappropriate grade-for-age were computed by the exposure variable and by key socio-demographic characteristics such as sex, ethnicity, household wealth, maternal education, slum of residence. Interaction terms between the main exposure and other covariates were assessed and stratified results are presented where appropriate.

### **11.3 Results**

Figure 22 shows the proportion of children enrolled in school at the various ages. A higher proportion of girls and boys get enrolled in primary school by age 6 in Viwandani than Korogocho slum. However, by age 10, about equal proportions of girls and boys in the two slums are enrolled in school. The proportion of girls and boys in both slums enrolled plateaus at just over 90% between the age of 10 and 13 years. After age 13, the proportion of children enrolled starts to drop, especially in Korogocho and for girls. Although children in the two slums tend to start school late, at age 14 more than 80% of children are still school.

Figure 22: Proportion of children enrolled in school at various ages

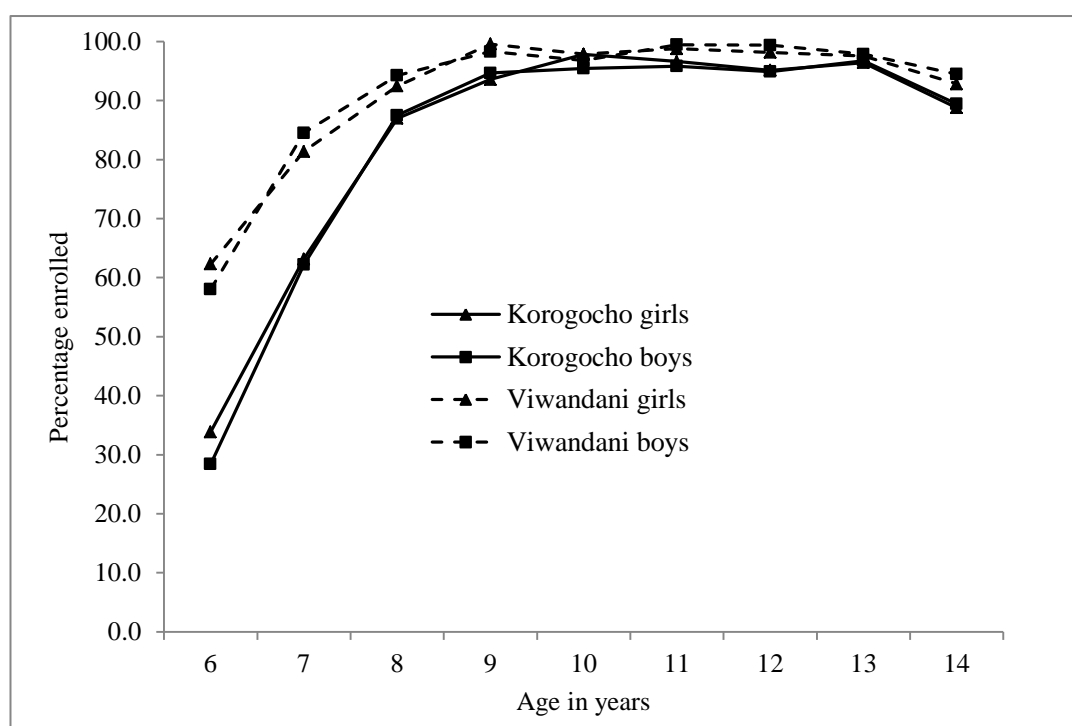


Table 52 shows proportions of children who had enrolled in school at age 7 or earlier and factors associated with timely enrolment, holding all other covariates constant. The adjusted results are presented for the whole group but also stratified on slum of residence.

About 71% of children aged 6 or 7 years were enrolled in school. About 81% of children in Viwandani had enrolled, compared with 65% in Korogocho; 77% of Kikuyu children were enrolled compared to 64% among the Luo and 58% among Cushites. While 79% of children whose mothers had secondary or higher education had enrolled in school, only 61% of children whose mothers had no formal education had enrolled. Lower proportions of children from households with a larger number of adults were in school by age 7.

Results from logistic regression models show that death of a mother in a household decreased the odds of a child's enrolling in school by age 7 by 44%. However, stratifying on slum of residence, only the children who had lost a father in Korogocho slum were significantly less likely to be enrolled. Children in both slums who lost a mother were less likely to be enrolled in school but on stratification this effect was insignificant at 95% confidence interval, a reflection of small number of maternal

orphans. After controlling for effect of other variables, boys had 11% lower odds of being enrolled in school compared with girls. Korogocho boys had 14% lower odds of being in school by age 7 than their female counterparts. Children from Viwandani slum had higher odds of being in school compared with those of Korogocho (aOR=1.87, p value <0.001), other factors being held constant. The odds of being enrolled in school were significantly lower in most other ethnic groups compared with the Kikuyu. The results further showed that children whose mothers had secondary or higher education had 47% higher odds of having enrolled in school compared with those whose mothers had no formal education. Household wealth appeared to be associated with the likelihood of enrolling in school by age 7 in the univariate models, but turned out to be insignificant after controlling for other covariates. A higher number of adults in the household were associated with lower odds of a child enrolling in school compared with those who lived in households with only one adult.



Table 52: Predictors of children having enrolled in school by age 7: results from logistic regression models

Variables	Number	Percent enrolled	Unadjusted OR (95% CI)	Adjusted (All) OR (95% CI)	Adjusted (Korogocho) OR (95% CI)	Adjusted (Viwandani) OR (95% CI)
Exposure						
Not exposed	6,030	71.2	1.00	1.00	1.00	1.00
Father died	106	66.0	0.78 (0.52,1.18)	0.68 (0.43,1.06)	0.57* (0.34,0.96)	1.24 (0.45,3.43)
Mother died	51	56.9	0.53* (0.30,0.93)	0.56* (0.32,1.00)	0.68 (0.34,1.33)	0.36 (0.13,1.00)
Other adult died	145	71.0	0.99 (0.69,1.42)	1.13 (0.78,1.64)	1.34 (0.88,2.06)	0.52 (0.23,1.14)
Age						
6 yrs	3,305	72.2	1.00	1.00	1.00	1.00
7 yrs	3,027	69.8	0.89* (0.80,0.99)	0.98 (0.88,1.10)	0.99 (0.86,1.13)	0.97 (0.79,1.19)
Sex						
Female	3,220	72.1	1.00	1.00	1.00	1.00
Male	3,112	70.0	0.90 (0.81,1.01)	0.89* (0.80,1.00)	0.86* (0.76,0.99)	0.96 (0.78,1.17)
Slum						
Korogocho	3,833	64.6	1.00	1.00		
Viwandani	2,499	80.9	2.32*** (2.06,2.62)	1.87*** (1.53,2.30)		
Ethnicity						
Kikuyu	1,939	77.4	1.00	1.00	1.00	1.00
Luhya	996	68.6	0.64*** (0.54,0.76)	0.61*** (0.51,0.73)	0.61*** (0.49,0.76)	0.61** (0.45,0.85)
Luo	1,415	63.5	0.51*** (0.44,0.59)	0.56*** (0.48,0.66)	0.55*** (0.46,0.66)	0.57*** (0.40,0.82)
Kamba	840	76.9	0.97 (0.80,1.18)	0.74** (0.60,0.91)	0.62** (0.45,0.85)	0.81 (0.61,1.08)
Cushites	661	58.4	0.41*** (0.34,0.49)	0.52*** (0.42,0.65)	0.52*** (0.41,0.66)	0.51* (0.27,0.93)
Other	481	79.8	1.16 (0.90,1.48)	0.82 (0.63,1.06)	0.76 (0.48,1.22)	0.84 (0.60,1.18)
Wealth						
Poorest	1,801	70.4	1.00	1.00	1.00	1.00
Middle	1,584	81.9	1.91*** (1.62,2.25)	1.20 (0.97,1.48)	--	1.18 (0.93,1.48)
Wealthiest	2,947	65.6	0.80*** (0.71,0.91)	1.12 (0.97,1.29)	1.16 (0.99,1.35)	0.88 (0.58,1.33)
Parental co-residence						
Both parents	4,377	71.7	1.00	1.00	1.00	1.00
Mother-HHH	757	68.3	0.85 (0.72,1.00)	0.74** (0.61,0.89)	0.78* (0.62,0.97)	0.62* (0.43,0.91)
Mother-Not HHH	185	62.2	0.65** (0.48,0.88)	0.53*** (0.39,0.73)	0.51*** (0.35,0.76)	0.57* (0.32,1.00)
Father only	428	74.3	1.14 (0.91,1.43)	1.32* (1.02,1.70)	1.27 (0.95,1.70)	1.43 (0.82,2.47)
None of parents	585	69.9	0.92 (0.76,1.11)	0.97 (0.78,1.21)	1.02 (0.80,1.31)	0.85 (0.55,1.30)
Maternal education						

None	751	61.3	1.00	1.00	1.00	1.00
Primary	3,736	70.1	1.49*** (1.26,1.75)	1.08 (0.88,1.31)	1.06 (0.85,1.31)	1.09 (0.66,1.81)
Secondary/higher	1,422	78.8	2.35*** (1.93,2.85)	1.47*** (1.17,1.84)	1.50** (1.15,1.96)	1.42 (0.84,2.41)
Unknown	423	70.5	1.51** (1.17,1.95)	0.99 (0.74,1.34)	1.04 (0.74,1.47)	0.88 (0.46,1.68)
Number of adults in HH						
1	987	73.7	1.00	1.00	1.00	1.00
2	3,243	71.8	0.91 (0.77,1.07)	0.76** (0.63,0.92)	0.80* (0.64,0.99)	0.65* (0.45,0.92)
3	1,108	72.0	0.92 (0.76,1.12)	0.82 (0.66,1.01)	0.81 (0.63,1.04)	0.79 (0.52,1.19)
4	524	66.4	0.71** (0.56,0.89)	0.71** (0.55,0.91)	0.77 (0.58,1.03)	0.52* (0.31,0.86)
5	470	63.2	0.61*** (0.49,0.78)	0.71* (0.55,0.92)	0.69* (0.52,0.93)	1.01 (0.50,2.02)
Total	6,332	71.0				

Stratified on slum due to significant interaction between exposure and slum of residence

Likelihood-ratio test  $\chi^2(3) = 7.54$ ; p value = 0.0564. Assumption: Model without interaction nested in one with interaction.

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$

Table 53 presents proportions of children who had ever repeated a grade and factors associated with repeating a grade. Overall, 12.2% of all children who had ever attended school had repeated a grade. About 13% of children who lost a mother repeated at least one grade while 12.2% of children who lived in households that did not experience an adult death repeated a grade. Higher proportions of children aged 9-11 years (16%), from Korogocho slum (15%), Cushites (17%), and those whose mothers had no formal education (17%) ever repeated a grade.

Results from the adjusted model show that children who were exposed to an adult death did not have significantly higher odds of having repeated a grade compared with those who did not lose an adult in their household. However, after stratification and controlling for other covariates, death of a mother in Viwandani slum was significantly associated with about 3.1 higher odds of having repeated a grade as compared with non-exposed children.

Age was associated with likelihood of having repeated a grade. Before stratification on slum, the results in the adjusted model show that Viwandani slum residence was associated with 55% lower odds of having repeated a grade as compared with Korogocho residence. Wealth was associated with children's repeating a grade in Korogocho slum, with children from the wealthiest households had 26% lower odds of having repeated a grade compared with the poorest. Maternal education was weakly associated with the likelihood of having repeated a grade in Korogocho, but not Viwandani. Children from Korogocho whose mothers had secondary or higher education had 28% lower odds of having repeated a grade compared with those whose mothers had no formal education. Child's sex, ethnicity, number of adults in household and parental co-residence were not significantly associated with the likelihood of having repeated a grade.

Table 53: Predicators of grade repetition: results from logistic regression models

Variables	Number	Percent repeated	Unadjusted OR (95% CI)	Adjusted (All) OR (95% CI)	Adjusted (Korogocho) OR (95% CI)	Adjusted (Viwandani) OR (95% CI)
Exposure						
Not exposed	9,672	12.2	1.00	1.00	1.00	1.00
Father died	187	7.5	0.58 (0.34,1.01)	0.57 (0.32,1.02)	0.53 (0.27,1.05)	0.76 (0.26,2.26)
Mother died	116	12.9	1.07 (0.62,1.85)	0.96 (0.55,1.67)	0.66 (0.33,1.33)	3.09* (1.22,7.84)
Other adult died	311	15.4	1.32 (0.96,1.80)	1.14 (0.83,1.58)	1.22 (0.87,1.72)	0.61 (0.21,1.72)
Age group						
6-8yrs	2,677	11.6	1.00	1.00	1.00	1.00
9-11yrs	3,248	16.0	1.45*** (1.25,1.69)	1.57*** (1.35,1.84)	1.49*** (1.24,1.79)	1.76*** (1.31,2.37)
12-14yrs	4,361	9.8	0.83* (0.71,0.97)	1.55*** (1.24,1.94)	1.51** (1.17,1.95)	1.57 (0.97,2.53)
Sex						
Female	5,231	12.2	1.00	1.00	1.00	1.00
Male	5,055	12.3	1.01 (0.90,1.13)	0.99 (0.87,1.11)	1.01 (0.87,1.16)	0.94 (0.74,1.19)
Slum						
Korogocho	6,360	14.9	1.00			
Viwandani	3,926	7.9	0.49*** (0.43,0.56)	0.45*** (0.35,0.57)		
Ethnicity						
Kikuyu	3,437	12.0	1.00	1.00	1.00	1.00
Luhya	1,516	11.3	0.93 (0.77,1.13)	0.84 (0.69,1.03)	0.82 (0.65,1.03)	0.96 (0.65,1.42)
Luo	2,221	12.7	1.06 (0.91,1.25)	0.88 (0.75,1.05)	0.84 (0.69,1.01)	1.21 (0.81,1.82)
Kamba	1,257	9.1	0.73** (0.59,0.91)	0.86 (0.68,1.09)	0.92 (0.66,1.30)	0.90 (0.64,1.25)
Cushites	1,122	16.8	1.48*** (1.23,1.79)	1.09 (0.87,1.36)	1.07 (0.84,1.36)	1.07 (0.50,2.29)
Other	733	11.7	0.97 (0.76,1.25)	1.24 (0.95,1.62)	1.18 (0.76,1.82)	1.32 (0.92,1.90)
Wealth						
Poorest	2,937	13.9	1.00	1.00	1.00	1.00
Middle	2,600	7.9	0.53*** (0.44,0.63)	0.98 (0.76,1.25)	--	1.07 (0.81,1.40)
Wealthiest	4,749	13.5	0.96 (0.84,1.10)	0.76*** (0.65,0.88)	0.74*** (0.63,0.86)	1.18 (0.71,1.99)
Parental co-residence						
Both parents	6,008	12.5	1.00	1.00	1.00	1.00
Mother-HHH	1,431	12.1	0.97 (0.81,1.15)	0.93 (0.77,1.14)	0.91 (0.73,1.13)	1.00 (0.66,1.51)
Mother-Not HHH	241	11.2	0.89 (0.59,1.33)	0.84 (0.55,1.28)	0.86 (0.53,1.40)	0.74 (0.32,1.75)
Father only	763	12.2	0.98 (0.78,1.23)	0.98 (0.76,1.27)	1.00 (0.75,1.32)	0.88 (0.49,1.57)
None of parents	1,843	11.6	0.92 (0.79,1.09)	1.00 (0.81,1.22)	0.97 (0.77,1.22)	1.08 (0.69,1.70)

Maternal education						
None	1,050	17.0	1.00	1.00	1.00	1.00
Primary	5,884	12.5	0.70*** (0.58,0.83)	0.95 (0.77,1.17)	0.91 (0.73,1.14)	1.60 (0.78,3.25)
Secondary/higher	2,008	9.6	0.52*** (0.42,0.65)	0.79 (0.61,1.02)	0.72* (0.53,0.97)	1.42 (0.68,2.95)
Unknown	1,344	11.2	0.62*** (0.49,0.78)	0.89 (0.67,1.19)	0.82 (0.60,1.13)	1.65 (0.72,3.80)
Grade category						
Lower primary	5,554	15.3	1.00	1.00	1.00	1.00
Higher primary	4,457	9.0	0.55*** (0.48,0.62)	0.42*** (0.35,0.51)	0.40*** (0.32,0.50)	0.50*** (0.34,0.74)
Secondary	275	1.5	0.08*** (0.03,0.22)	0.06*** (0.02,0.17)	0.04*** (0.01,0.18)	0.11** (0.03,0.48)
Number of adults in HH						
1	1,570	12.2	1.00	1.00	1.00	1.00
2	4,092	11.0	0.89 (0.74,1.06)	0.90 (0.74,1.09)	0.88 (0.70,1.11)	0.93 (0.63,1.37)
3	2,250	12.7	1.05 (0.86,1.27)	1.13 (0.91,1.39)	1.13 (0.89,1.44)	1.12 (0.73,1.72)
4	1,220	12.7	1.04 (0.83,1.31)	1.09 (0.86,1.39)	1.17 (0.89,1.54)	0.82 (0.47,1.41)
5+	1,154	14.8	1.25* (1.00,1.56)	1.21 (0.95,1.55)	1.14 (0.87,1.50)	1.72 (0.99,2.99)
Total	10,286	12.2				

Stratified on slum due to significant interaction between exposure and slum of residence

Likelihood-ratio test chi2 (3)= 6.74; p value= 0.0807. Assumption: Model without interaction nested in one with interaction.

\*p<0.05, \*\* p<0.01, \*\*\* p<0.001

Table 54 presents descriptive results for interruption of schooling in successive calendar years. The percentages indicate the proportions of children who had ever been to school that had interrupted schooling for at least one calendar year. Overall, the lowest proportion of children who interrupted schooling was in 2003 (10%) and highest in 2006 (15%). In all the five years observed, higher proportions of children from Korogocho slum interrupted schooling compared with Viwandani. Also in all the years, higher proportions of children from the Luo and Cushitic ethnic backgrounds interrupted schooling compared with other ethnicities. Higher proportions of children who lived with a mother who was not the household head and no father or whose mothers had no formal education interrupted schooling compared with their counterparts.

Table 55 shows results from random effects logistic regression models for factors associated with the interruption of children's schooling. From the unadjusted and adjusted models, it can be seen that experiencing an adult death was not significantly associated with interruptions in children's schooling. The un-stratified adjusted full model showed that children in Viwandani were about 40% less likely to interrupt schooling as compared with children in Korogocho slum. Children from Korogocho whose mother had died had 54% lower odds of interrupting schooling than other children, while those of Viwandani had 3.7 times higher odds compared with the unexposed children.

Ethnicity was not significantly associated with school interruption, although the odds of interrupting were higher for ethnicities other than Kikuyu ethnic group. On stratifying by slum of residence, among children from Korogocho, the Luo and Cushitic ethnic backgrounds had significantly higher odds of having their schooling interrupted compared with the Kikuyu children while those from Viwandani had lower but insignificant odds of interruptions in their schooling. Household wealth status and child's sex were not significantly associated with the likelihood of interrupting school. In both unadjusted and adjusted models, children who lived with neither parent were more likely to experience interruptions in their schooling compared with those who lived with both parents. The direction of the association remained the same upon

stratification but with children who lived in Viwandani having higher adjusted odds (OR=2.6; p value<0.01) compared to Korogocho (OR=1.7; p value< 0.01).

Unadjusted and adjusted results show that age group 9-11 was associated with lower odds of interruptions in their schooling in both slums compared with age category 6-8 years. For older children (12-14 years), however, the direction of association depends on the slum of residence. Children aged 12-14 years resident in Korogocho had lower adjusted odds (OR=0.74; p value<0.05) of interruptions in their schooling compared with those aged 6-8 years, while for Viwandani, children aged 12-14 years had higher adjusted odds (OR=1.74; p value <0.05) of interruptions in their schooling compared with those aged 6-8 years. Neither maternal education nor number of adults in a household was a significant predictor of whether children's schooling was interrupted.

Table 54: Proportions of children who ever-schooled but interrupted schooling in respective calendar years

Variables	2003		2004		2005		2006		2007	
	% not in school	Total	% not in school	Total	% not in school	Total	% not in school	Total	% not in school	Total
Exposure										
Not exposed	13.1	5,562	10.3	5,911	13.7	6,470	14.7	6,711	13.5	7,107
Father died	17.8	45	11.4	79	15.0	133	13.3	151	14.9	141
Mother died	0.0	16	10.6	47	7.4	68	13.8	94	15.8	76
Other adult died	14.6	82	10.9	147	12.3	203	14.8	264	12.6	223
Sex										
Female	12.2	2,909	9.5	3,164	13.5	3,500	14.3	3,700	13.8	3,837
Male	14.0	2,796	11.1	3,020	13.8	3,374	15.0	3,520	13.2	3,710
Slum										
Korogocho	14.7	3,750	10.7	3,976	15.9	4,392	17.6	4,893	16.1	4,521
Viwandani	10.0	1,955	9.6	2,208	9.7	2,482	8.4	2,327	9.6	3,026
Ethnicity										
Kikuyu	9.4	2,040	7.3	2,162	10.3	2,302	11.5	2,275	11.3	2,363
Luhya	15.5	788	11.6	882	14.5	999	16.6	1,106	14.8	1,116
Luo	16.1	1,206	13.2	1,297	15.6	1,471	17.3	1,730	15.8	1,657
Kamba	13.7	593	10.2	670	12.6	788	11.7	784	12.1	995
Cushites	16.7	744	13.3	783	20.1	849	20.0	849	17.6	852
Other	10.2	334	8.5	390	12.0	465	10.3	476	9.6	564
Wealth										
Poorest	14.2	1,644	10.4	1,759	13.5	1,951	14.6	2,096	14.1	2,154
Middle	9.5	1,344	9.6	1,497	9.3	1,665	8.3	1,527	8.7	1,957
Wealthiest	14.2	2,717	10.6	2,928	15.9	3,258	17.4	3,597	15.9	3,436
Parental co-residence										
Both parents	13.5	2,877	10.2	3,374	13.6	3,970	15.3	4,463	14.1	4,985
Mother-HHH	11.0	812	10.0	871	12.5	973	15.7	1,006	13.2	994
Mother-Not HHH	17.9	123	19.1	136	20.1	159	18.8	186	16.8	203
Father only	13.5	430	8.0	477	11.0	547	11.8	526	12.0	499
None of parents	12.9	1,463	10.7	1,326	15.0	1,225	11.5	1,039	10.7	866
Maternal education										
None	19.2	610	14.4	682	21.7	822	19.55	880	16.3	888
Primary	13.2	3,179	10.8	3,498	13.3	3,905	15.6	4,200	14.0	4,386



Secondary/higher	12.0	946	9.2	1,104	10.5	1,289	11.03	1,414	11.4	1,606
Unknown	10.2	970	6.7	900	12.0	858	10.19	726	11.8	667
Age category										
6-8	23.8	2,281	19.0	2,528	23.9	2,902	30.5	2,872	31.2	2,932
9-11	3.3	1,865	2.1	1,959	3.1	2,050	3.38	2,365	2.2	2,617
12-14	9.2	1,559	6.8	1,697	9.3	1,922	5.09	1,983	2.4	1,998
Number of adults in HH										
1	12.2	1,462	11.4	879	13.6	976	14.73	1,154	11.5	1,070
2	14.2	1,998	11.0	2,438	15.5	2,778	17.33	3,053	14.38	3,429
3	12.5	1,116	9.3	1,355	11.6	1,539	10.83	1,496	12.73	1,532
4	12.3	600	11.1	782	12.5	826	12.95	757	11.69	753
5	13.6	529	7.7	730	12.1	755	12.89	760	15.73	763
Total	13.1	5,705	10.3	6,184	13.6	6,874	14.64	7,220	13.5	7,547

Table 55: Predictors of interrupting schooling: results from random effects regression models

Variables	Unadjusted OR (95% CI)	Adjusted (All) OR (95% CI)	Adjusted (Korogocho) OR (95% CI)	Adjusted (Viwandani) OR (95% CI)
Exposure				
Not exposed	1.00	1.00	1.00	1.00
Father died	0.98 (0.46,2.10)	1.14 (0.51,2.53)	1.06 (0.41,2.74)	1.34 (0.29,6.18)
Mother died	0.80 (0.27,2.38)	0.87 (0.29,2.60)	0.46 (0.10,2.06)	3.74 (0.66,21.12)
Other adult died	1.44 (0.86,2.42)	1.27 (0.76,2.13)	1.38 (0.81,2.38)	0.74 (0.15,3.59)
Sex				
Female	1.00	1.00	1.00	1.00
Male	0.92 (0.76,1.12)	0.93 (0.77,1.13)	1.03 (0.82,1.28)	0.75 (0.51,1.11)
Slum				
Korogocho	1.00	1.00		
Viwandani	0.58*** (0.47,0.72)	0.60** (0.41,0.87)		
Ethnicity				
Kikuyu	1.00	1.00	1.00	1.00
Luhya	1.14 (0.84,1.55)	1.23 (0.90,1.67)	1.27 (0.88,1.83)	1.04 (0.58,1.90)

Luo	1.36* (1.04,1.76)	1.26 (0.96,1.65)	1.44* (1.07,1.93)	0.64 (0.31,1.33)
Kamba	1.04 (0.74,1.46)	1.34 (0.95,1.90)	1.41 (0.83,2.37)	1.15 (0.69,1.90)
Cushites	1.27 (0.92,1.76)	1.31 (0.90,1.90)	1.48* (1.00,2.20)	0.68 (0.18,2.52)
Other	0.76 (0.48,1.20)	1.00 (0.63,1.61)	1.66 (0.82,3.35)	0.67 (0.34,1.30)
Wealth				
Poorest	1.00	1.00	1.00	1.00
Middle	0.67** (0.50,0.89)	0.97 (0.66,1.43)	--	0.87 (0.57,1.35)
Wealthiest	1.13 (0.90,1.41)	0.97 (0.76,1.24)	0.98 (0.76,1.26)	0.72 (0.27,1.91)
Parental co-residence				
Both parents	1.00	1.00	1.00	1.00
Mother-HHH	1.22 (0.91,1.62)	1.28 (0.94,1.74)	1.29 (0.90,1.83)	1.19 (0.62,2.26)
Mother-Not HHH	1.75 (0.99,3.07)	1.76 (1.00,3.11)	1.72 (0.89,3.32)	2.04 (0.66,6.33)
Father only	1.08 (0.74,1.59)	1.02 (0.68,1.55)	0.97 (0.61,1.55)	1.22 (0.49,3.04)
None of parents	1.88*** (1.49,2.37)	1.84*** (1.37,2.46)	1.65** (1.18,2.30)	2.56** (1.39,4.71)
Maternal education				
None	1.00	1.00	1.00	1.00
Primary	1.02 (0.74,1.41)	1.10 (0.76,1.59)	1.11 (0.75,1.63)	1.16 (0.40,3.38)
Secondary/higher	0.78 (0.53,1.14)	0.94 (0.61,1.44)	1.00 (0.62,1.62)	0.90 (0.30,2.73)
Unknown	1.40 (0.96,2.05)	1.06 (0.67,1.68)	1.28 (0.79,2.09)	0.61 (0.17,2.12)
Age category				
6-8	1.00	1.00	1.00	1.00
9-11	0.30*** (0.23,0.39)	0.27*** (0.21,0.35)	0.26*** (0.19,0.35)	0.29*** (0.16,0.52)
12-14	1.09 (0.89,1.34)	0.94 (0.76,1.17)	0.74* (0.58,0.96)	1.74* (1.14,2.66)
Number of adults in HH				
1	1.00	1.00	1.00	1.00
2	0.96 (0.74,1.26)	1.09 (0.83,1.45)	1.07 (0.77,1.47)	1.28 (0.72,2.27)
3	0.95 (0.70,1.28)	1.02 (0.75,1.40)	0.87 (0.61,1.26)	1.57 (0.84,2.94)
4	1.09 (0.77,1.54)	1.13 (0.79,1.61)	0.99 (0.66,1.49)	1.70 (0.81,3.58)
5	0.94 (0.65,1.37)	0.91 (0.62,1.35)	0.86 (0.56,1.32)	1.20 (0.45,3.21)

Number of observations =29,708; Number of groups =10,499; Rho=0.38

\* p<0.05, \*\* p<0.01, \*\*\* p<0.001

Table 56 shows the percentage distribution of children who were in a lower grade for their age and factors associated with inappropriate grade-for-age. Overall, about 75.2% of children who ever enrolled in school were in a lower grade for their age when last observed. About 88% of children who had lost a mother were not in the right grade for their age compared with 75% of un-exposed children. About 81% of children in Korogocho were in an inappropriate grade-for-age compared with 65% in Viwandani. About 77% of boys were in an inappropriate grade for their age compared with 74% of girls. Luo, Cushites, children who lived with neither parents, those born to mothers with no formal education, and those who lived in household with 3 or more adults were all more likely to be in an inappropriate grade-for-age than their counterparts.

Unadjusted estimates showed that children who lost a mother or other non-parental adult in a household were more likely to lag behind in school than those who lived in households that had not experienced an adult death. After controlling for other covariates, however, only children who lost a mother had significantly higher odds of lagging behind (OR=2.1; p value<0.05). Before stratifying on slum of residence, residing in Viwandani was associated with 54% lower odds of being of an inappropriate grade-for-age. Stratifying on slum of residence showed that the effect of a maternal death on a child's school progression was only important in Viwandani slum, where the odds of being of inappropriate grade-for-age was about 6.4 times higher than that of non-exposed children (aOR=6.35; p value<0.01). It should be noted that, although non-significant, death of a mother in Korogocho was also associated with higher odds of a child lagging behind in school. In both unadjusted and adjusted models, children aged 9 years or more had an increased likelihood of being in an inappropriate grade for their age. The Mantel-Haenszel estimation for trend showed a significant trend with the odds of being of inappropriate grade-for-age increasing by approximately 2.6 times (p value <0.0001) for a unit increase in age category. Boys were at least 20% more likely to be in an inappropriate grade-for-age than girls in both slums. For all identifiable ethnic groups, the odds of Kikuyu children being in an inappropriate grade for their age were lower than children from other ethnic backgrounds. Children born to mothers with secondary or higher education were less likely to be in an inappropriate grade-for-age. In models stratified on slum, the association was only significant for Korogocho.

Table 56: Predictors of inappropriate grade-for-age: results from logistic regression models

Variables	Number	Percent Not right age- for-grade	Unadjusted OR (95% CI)	Adjusted (All) OR (95% CI)	Adjusted (Korogocho) OR (95% CI)	Adjusted Viwandani OR (95% CI)
Exposure						
Not exposed	9,648	74.9	1.00	1.00	1.00	1.00
Father died	190	75.8	1.05 (0.75,1.47)	0.91 (0.62,1.34)	0.96 (0.57,1.63)	0.85 (0.47,1.52)
Mother died	121	87.6	2.37** (1.38,4.08)	2.13* (1.19,3.80)	1.37 (0.73,2.59)	6.35** (1.81,22.22)
Other adult died	317	80.1	1.35* (1.02,1.79)	1.10 (0.82,1.49)	1.01 (0.71,1.43)	1.33 (0.73,2.43)
Age						
6-8 yrs	2,534	49.2	1.00	1.00	1.00	1.00
9-11 yrs	3,139	78.6	3.80*** (3.38,4.26)	3.73*** (3.31,4.20)	3.23*** (2.75,3.80)	4.43*** (3.71,5.29)
12-14 yrs	4,603	87.2	7.01*** (6.24,7.87)	7.31*** (6.43,8.32)	7.01*** (5.87,8.37)	7.73*** (6.38,9.37)
Sex						
Female	5,229	73.9	1.00	1.00	1.00	1.00
Male	5,047	76.6	1.16** (1.06,1.27)	1.22*** (1.11,1.35)	1.20** (1.05,1.38)	1.24** (1.07,1.43)
Slum						
Korogocho	6,351	81.4	1.00	1.00		
Viwandani	3,925	65.1	0.43*** (0.39,0.47)	0.46*** (0.39,0.55)		
Ethnicity						
Kikuyu	3,433	71.1	1.00	1.00	1.00	1.00
Luhya	1,514	78.7	1.50*** (1.30,1.73)	1.76*** (1.50,2.06)	1.69*** (1.37,2.08)	1.90*** (1.49,2.42)
Luo	2,219	81.0	1.74*** (1.53,1.98)	1.69*** (1.46,1.95)	1.69*** (1.42,2.01)	1.71*** (1.31,2.24)
Kamba	1,257	68.3	0.88 (0.76,1.01)	1.39*** (1.18,1.63)	1.68** (1.20,2.36)	1.33** (1.09,1.61)
Cushites	1,120	85.1	2.32*** (1.94,2.78)	1.68*** (1.35,2.09)	1.57*** (1.22,2.01)	2.32** (1.40,3.85)
Other	733	66.3	0.80* (0.68,0.95)	1.33** (1.09,1.62)	1.91** (1.18,3.10)	1.23 (0.98,1.54)
Wealth						
Poorest	2,933	75.3	1.00	1.00	1.00	1.00
Middle	2,598	66.0	0.64*** (0.57,0.71)	1.03 (0.88,1.20)	--	1.03 (0.87,1.21)
Wealthiest	4,745	80.2	1.32*** (1.19,1.48)	0.87 (0.76,1.00)	0.87 (0.75,1.01)	0.95 (0.69,1.32)
Parental co-residence						
Both parents	6,005	72.7	1.00	1.00	1.00	1.00
Mother-HHH	1,435	77.7	1.31*** (1.14,1.50)	1.08 (0.92,1.28)	1.20 (0.96,1.49)	0.96 (0.74,1.23)
Mother-Not HHH	241	72.6	1.00 (0.75,1.33)	1.26 (0.91,1.75)	1.57 (0.98,2.50)	0.99 (0.62,1.59)
Father only	761	78.3	1.36** (1.13,1.63)	1.06 (0.85,1.32)	1.21 (0.91,1.61)	0.88 (0.62,1.25)

None of parents	1,834	80.4	1.54*** (1.36,1.75)	0.98 (0.82,1.18)	1.04 (0.83,1.30)	0.92 (0.69,1.23)
Maternal education						
None	1,050	83.1	1.00	1.00	1.00	1.00
Primary	5,877	76.0	0.65*** (0.54,0.77)	0.82 (0.67,1.01)	0.74* (0.58,0.95)	1.17 (0.79,1.75)
Secondary/higher	2,007	66.8	0.41*** (0.34,0.49)	0.65*** (0.52,0.82)	0.55*** (0.41,0.73)	0.98 (0.65,1.48)
Unknown	1,342	78.1	0.73** (0.59,0.89)	0.61*** (0.47,0.79)	0.55*** (0.40,0.77)	0.88 (0.54,1.43)
Number of adults in HH						
1	1,562	75.4	1.00	1.00	1.00	1.00
2	4,071	70.9	0.79*** (0.70,0.91)	1.03 (0.88,1.21)	1.08 (0.87,1.34)	0.96 (0.75,1.21)
3	2,240	76.8	1.08 (0.93,1.25)	0.95 (0.80,1.14)	0.96 (0.75,1.21)	0.93 (0.71,1.22)
4	1,225	79.1	1.23* (1.03,1.48)	0.91 (0.74,1.12)	0.89 (0.68,1.16)	0.94 (0.68,1.30)
5	1,178	82.6	1.55*** (1.28,1.87)	0.92 (0.74,1.15)	1.02 (0.78,1.34)	0.73 (0.50,1.06)
Total	10,276	75.2				

Stratified on slum even though interaction between exposure and slum of residence was not significant

Likelihood-ratio test  $\chi^2(3) = 6.79$ ; p value = 0.0787. Assumption: Model without interaction nested in one with interaction.

\*  $p < 0.05$ , \*\*  $p < 0.01$ , \*\*\*  $p < 0.001$

## 11.4 Discussion

This chapter explores the relationship between death of an adult in a household and children's educational attainment. In total, four schooling outcomes were examined: school enrolment for children aged 7 or less; grade repetition; interruption of schooling; and grade-for-age.

The results show that the effect of an adult's death on children's schooling depended on the adult's relationship with the child and the type of outcome being examined, but also on whether a child lived in either Viwandani or Korogocho slum. Viwandani children who lost a mother generally had poorer educational attainment than other children in the slum. They were less likely to be enrolled in school by age 7, more likely to repeat grades, more likely to interrupt schooling (not significant) and more likely to be of an inappropriate grade-for-age as at the end of observation. For Korogocho slum, death of a mother was not associated with significantly poorer educational attainment. In fact, for some outcomes, such as grade repetition and interruption of schooling, the association was in the opposite direction to that in Viwandani. While fathers often provide material and financial support to children, they might have a limited role in the care and emotional support to their children to stay in school. But it might also be that, when a father dies, the mother steps up efforts to ensure that her children stay in school, for example, by getting into "employment" and drawing on support from relatives and friends for fees and other scholastic materials.

These findings are broadly in line with those of other studies that have shown that orphaned children, particularly maternal orphans, are more likely to have poorer educational attainment than their non-orphaned counterparts <sup>[5, 18, 22, 23, 25, 281]</sup>. While this is the general trend, other studies have only demonstrated this finding in only low socio-economic status households. Timaeus and Boler found, in a study carried out in South Africa, that paternal orphanhood rather than maternal orphanhood was associated with poorer educational attainment net of household socio-economic status <sup>[26, 351]</sup>. The fact that studies from a range of populations have shown that orphanhood has a marked, little or no impact on children's schooling brings the local context into the picture.

The striking finding that has arisen from these analyses and has not been fully explained is why the educational attainment of children from Viwandani who lost a mother is worse than that of other children, yet the same was not true in Korogocho? This is despite the finding that Viwandani slum children have better educational attainment overall compared with Korogocho. This finding takes us back to the literature on educational attainment and orphanhood. While in majority of studies, negative schooling outcomes have been observed for orphaned children, in some studies no differences were observed between orphaned and no-orphaned children <sup>[305, 352]</sup>. Also, variations in educational attainment have been noted, depending on what aspect of schooling is measured. For example Ainsworth et al. showed that orphaned children delayed enrolment into school but were not more likely to drop out than non-orphans <sup>[5]</sup>. These differences have contributed to the ongoing debate, as a possible explanation, on how different communities cope with the effects of adult death, particularly due to HIV/AIDS, and how the coping mechanisms and child caring practices may impact on child well-being in general. The role of the extended family in caring for orphans in Africa has been well documented. While there are marked variations in orphan caring practices across Africa, generally paternal orphans tend to live with their surviving mothers, while maternal orphans often to live with relatives other than their surviving father <sup>[25]</sup>. Sisters and brothers to the deceased adult often take up orphans if the surviving parent is unable to look after them or if both parents have died, as is often the case with HIV/AIDS related deaths.

In general, placement of orphans may be a highly selective process with orphaned children most likely to be taken up by close relatives such as aunts and uncles or grandparents, who may even be economically better off than were the children's parents. As a result, any impact of orphanhood might be fully compensated for and cancelled out in some households or communities. Indeed, Ardington, Leibbrandt et al. showed that, while the effects of orphanhood were expected to strengthen over time owing to increasing adult mortality due to HIV/AIDS in South Africa, this was not the case. Rather what shifted over the period was increasing involvement of grandparents in child care. It should be noted, however, that the socio-economic situation in South Africa differs from that of most countries in sub-Saharan Africa. South Africa provides a non-contributory pension scheme to all elderly individuals. As a result, some

grandparents may be financially more capable of looking after their grandchildren than the parents. But still, even with the placement of orphans in economically better households, it is argued that orphaned children may have poor schooling outcomes than non-orphans with whom they live in the same households probably<sup>[18]</sup>. This implies that living in an economically better household does not always translate into better educational attainment for orphans probably due to discrimination at household level or emotional distress associated with bereavement<sup>[18]</sup>.

Differences in the coping mechanisms and the availability of social safety nets between the various populations may partly explain the differing educational attainment seen in various studies in sub-Saharan Africa of the effects of orphanhood. In the current political dispensation in Kenya, school fees have been removed and are no longer a barrier to accessing education. While Viwandani and Korogocho are both slums, the two are quite different demographically and economically. The population in Korogocho is more settled, households are larger (average of 3 members per household), adult residents are mainly self-employed in petty trade, and are poorer and less educated. Viwandani slum has a generally younger population, with smaller households (average of 2.3 members per household). Its inhabitants tend to be more educated with a sizeable proportion in salaried employment in the nearby industrial estate<sup>[133]</sup>. Based on these differences, one can speculate that, although Viwandani slum households are better endowed economically, Korogocho slums may have better resilience and social capital to draw on in times of need.

In this study, boys were less likely to be enrolled in school by age 7 and they were more likely to be in an inappropriate grade for their age than girls, while there was no significant gender difference in risk of interruption of schooling or repeating a grade. Gender differences in educational attainment have been of interest and have been highlighted in a number of research papers where girls are often shown to have poorer schooling outcomes<sup>[23, 352]</sup>. However, studies in other settings in sub-Saharan Africa have also found that girls have a schooling advantage compared with boys<sup>[26, 351]</sup>. In some cultures, pronounced child gender preferences often exist with more investment including education being put into boys. Girls are seen as having a poor return on investment as they tend to marry and leave the family. Also, children's gender roles at a



household level might affect their schooling, for example girls might be more involved in household chores like washing up, cooking, caring for the sick and looking after their younger siblings. And, importantly, traditional chores for boys such as herding small stock, scaring birds off crops and so on are not common in an urban context. But in this population, the challenges for boys are different and probably are due to the social influences in the slums. Boys tend to be involved in income generation to support family at an early stage, truancy, alcohol abuse, day-time video shows, street begging and other criminal gang activities.

Children from the Kikuyu ethnic background have better schooling outcomes than the other ethnicities except for grade repetition and interruption of schooling, where there are no differences. In Kenya, ethnicity is an important predictor of both child and adult health and social outcomes. While these differences may be partly explained by differences in cultures, they also reflect socio-economic inequalities between the various tribes. The Kikuyu are generally more socio-economically empowered and this may translate into the better health and schooling outcomes for their children. In South Africa, where the socio-economic inequality is mainly grounded in race, it is often observed that black children have poorer educational outcomes than other races that are socio-economically more endowed <sup>[353, 354, 355]</sup>. It should be noted, however, that, because of the way household socio-economic status was measured in this study, it is not possible to capture short-term financial challenges. The wealth index fails to capture and adequately control for the socio-economic differences that may affect children in the different subgroups. Maternal education not only translates into better child care knowledge and survival, it also impacts on economic opportunities for mothers and thus their children's wellbeing <sup>[315, 356]</sup>. Findings here showed that secondary or higher maternal education was associated with greater enrolment in school by age 7 and a lower likelihood of being in an inappropriate grade-for-age. These findings are consistent with other child wellbeing outcomes such as child survival, where higher maternal education has been found to be associated with better health outcomes.

Research over the years has shown that, in the slums, children have been driven out of public schools to private informal schools, the so called "private schools for the poor", due to inadequate supply of public schools in the slums and perceived poor quality due

to overcrowding <sup>[208, 357]</sup>. The increase in enrolment upon the implementation of the FPE policy in 2003 with limited corresponding increase in available public schools and teachers put a strain on the quality of school provision characterised by overcrowding, high pupil-teacher ratio and poor scores in national examinations. While the Free Primary Education (FPE) policy takes away the burden of paying school fees to access school, other barriers to schooling remain such as availability of nearby schools, quality of schools, and family support for children's schooling.

Previous studies have looked at one or two education attainment outcomes, however this study explored a range of outcomes to obtain a fuller understanding of the impact of losing an adult on child schooling. The importance of examining a range of educational outcomes is that, as we have seen here, the impact of the exposure may be different for the various educational outcomes. Secondly, the various facets of educational challenges (enrolment, grade repetition, grade-for-age and interruption) occur to varying degrees and their respective impact on overall school achievement is also likely to be varied, hence the need to have a fuller understanding of these processes whenever the data allow this. Some studies have limited their assessment on summary measures such enrolment or years of schooling. If enrolment is high as a result of government interventions such as free primary education, deciphering effects due to household-level factors such as orphanhood may be difficult as there is limited variability in the outcome measure <sup>[18, 281]</sup>. Indeed in this study, preliminary analyses showed that most children attend school at one time or another even if it just for a term or a single academic year. It was for this reason that analysis of late enrolment was limited to children who were aged 7 and below.

### **Policy implications and future research**

Should maternal orphans in Viwandani be targeted? Some earlier studies suggest that orphans should not be targeted but rather that interventions should be directed at reducing socio-economic inequalities in general <sup>[352]</sup>, while other authors support targeting orphans <sup>[23]</sup>. While results here clearly show that maternal orphans in Viwandani are disadvantaged, it is important to appreciate that Korogocho slum has worse outcomes overall probably because of its poorer socio-economic status. Besides, earlier studies have indicated a general schooling disadvantage to slum children in

comparison with the general population <sup>[350, 358]</sup>. All these considerations highlight the importance of socio-economic inequality as a major obstacle to schooling in slums and therefore a more holistic approach to improving slum education might be a better approach than targeting only orphans in the slums. Some researchers have argued that, in the context of wide spread poverty, targeting orphans will not only be unfair but also ineffective in raising school participation of orphans and non-orphaned children <sup>[22]</sup>. More research relating orphanhood to actual individual child school performance is needed, with probably, a better control for household socio-economic status. Also more research, probably anthropological in design, should be carried out to unravel and contextualise the schooling disadvantage of orphaned children in Viwandani. Finally, since a sizeable proportion of children enrol in school late, there is need to explore why this is the case. This might give further insights on how to improve education in slums.

### **Data quality and limitations**

Information on parental survivorship for children who are born outside of the surveillance area is not collected and remains unknown. When survivorship of parents of such children is not accounted for, the level of orphanhood and its effects are underestimated. Other important determinants of school participation such as quality of schools, and distance to school were not controlled for and this leaves an incomplete picture. The data used here were derived from a longitudinal surveillance system in which migration in and out of the slums is common. It is possible that attrition, if related to unmeasured determinants of child educational attainment, could bias the estimates obtained here. Poverty, especially sudden changes in household income might be related to both parental survival and child education but was not properly accounted for before and after death of an adult as socio-economic data are not collected regularly.

### **Conclusions**

Adult death negatively impacts child educational attainment, particularly that of maternal orphans in Viwandani slum. These orphans were more likely to repeat a grade and be of an inappropriate grade-for-age. Thus, the effects of adult deaths seem to be setting specific. Socio-demographic factors such as gender and ethnicity and household level factors such as maternal education and parental co-residence are also important predictors of child educational attainment.

## **12. Discussion**

### **12.1 Introduction**

This thesis is about how adult deaths impact children in affected households. The overall discussion chapter summarises key findings, highlights strengths and limitations and provides recommendations for policy and research based on the results in the various chapters. In order to understand how adult death in a household may impact children, the thesis aimed to examine three issues: i) the current adult mortality situation in the population, with a focus on levels, trends and causes of adult deaths; ii) the impact of adult death on children's circumstances in terms of where and with whom they live; and iii) lastly, children's health and social outcomes and how adult mortality may impact these outcomes. Children's household circumstances are represented by their migration experience and household living arrangements. Children's health and social outcomes are represented by their survival, health care utilisation (immunisation) and educational attainment.

Interpretations of individual results are not repeated here as these are already covered in detail in the discussion sections of the respective results chapters. The focus here is to highlight key findings and put them in a broader context discussing how they interrelate, their relevance to the wider Kenyan population and their programmatic implications. Strengths and limitations of the study design and analytical methodologies are discussed for each aim and, where applicable, proposals are made on how to better handle similar studies in the future. Lastly, conclusions and summary of key policy recommendations are proposed, focusing on findings that are amenable to intervention. Recommendations are also made on potential future research priorities based on insights from the results.

### **12.2 Levels, trends and causes of adult deaths:**

The first aim was to describe and estimate levels, trends and causes of adult death in Nairobi city informal settlements. Adult mortality in the two informal settlements studied is high, particularly among women, and as such life expectancy at 15 years is correspondingly low. Significant differentials in the risk of adult death, net of other

factors that were measured, include; gender, slum of residency, ethnicity, wealth status and educational attainment. Women, residents of Korogocho slum, the Luo ethnic group, the poorest and those with no or little formal education had a higher risk of death than other groups. The survival advantage by slum was restricted to women, with Viwandani slum women having better survival than Korogocho slum women. Generally, in most populations, men have higher mortality than women. However, recent research in populations with generalised HIV/AIDS epidemics, has found that HIV/AIDS-related mortality tends to be higher among women of reproductive age than men <sup>[359, 360, 361]</sup>. Further discussion of the higher mortality among women is provided in more detail below. Additionally, it appears that differences in HIV burden by ethnicity and slum of residence translates into differential mortality by ethnicity and slum of residence <sup>[2, 12, 13]</sup>.

Overall, HIV/AIDS is the leading cause of death. Among females, HIV/AIDS, cancer and diabetes were the most important causes of death while, among men, the top three causes of death were; injuries, HIV/AIDS and tuberculosis in that order. Women and residents of Korogocho had a higher risk of death from HIV/AIDS than men and residents of Viwandani respectively. However, men had higher mortality from non-HIV/AIDS causes than women. The Luo ethnic group had a higher risk of death from HIV/AIDS than other ethnicities. Cultural practices such as wife inheritance and lack of circumcision are common among the Luo and have been identified as possible explanations for the high prevalence of HIV this ethnic group <sup>[12, 13, 237]</sup>. Women's higher risk of HIV infection is related to their biological vulnerability but also the unfavourable gender power relations to negotiate for safer sex <sup>[362, 363, 364]</sup>.

A steady decline in adult mortality occurred, especially among women over the study period. The decline in mortality was, however, only demonstrable for certain causes of death. For others, there was no trend, while in one there was an increase in mortality. The sharpest decline was observed for HIV/AIDS, while an increase in risk of death was observed for injuries especially among men. The decline in mortality from HIV/AIDS corresponds to improvements in the management of HIV/AIDS through suppression of the virus that causes AIDS using ART and better management of opportunistic infections that has taken place in the last decade <sup>[241, 365, 366]</sup>. The

downward trend in mortality over the years was more marked among residents of Korogocho slum. Women and Korogocho residents have a higher prevalence of HIV according to both previous studies <sup>[2, 234]</sup>, and the results of this thesis, which has also shown that these groups have a higher risk of death from HIV/AIDS in spite of the declining trend.

Non-communicable diseases are increasingly becoming important public health challenges in Kenya and the sub-Saharan Africa region <sup>[1, 248]</sup>. They are expensive to manage both for the affected individuals and their families and for the health-care system as a whole. Diabetes and cardiovascular diseases and their risk factors, such as obesity, hyperlipidemia and hypertension, are becoming increasingly common and yet they often go unnoticed. Previously these conditions were thought to be diseases of the wealthy and old age, but this is rapidly changing with many poor and young adults getting affected. Diabetes, cancer, cardiovascular diseases and injuries are among the top causes of death in this population, yet the Kenyan Ministry of Health does not have a clear and well-funded prevention and control policy for non-communicable diseases. Prevention and treatment efforts are isolated or *ad hoc*, not monitored or evaluated and, as such, limited resources and attention is given to this growing public health challenge.

### Strength and limitations:

Most sources of mortality data in sub-Saharan Africa do not provide information on specific causes of death. However, the DSS data used here collect cause-of-death information and this makes it possible to study at least the leading causes of death individually. The additional information on specific causes of adult death is crucial in understanding and informing planning of interventions and allocation of resources. It is also important in projecting and making assumptions about future mortality for a given population <sup>[361]</sup>. For example, population projections might need to account for HIV/AIDS mortality to be able to get an accurate prediction of future mortality trends. Unfortunately, data of this nature are rare and, where they are available, the follow-up is sometimes short or the numbers of individuals under surveillance are small.

The data used in this thesis span a period of 5 years of continuous surveillance generating prospectively collected information. The prospective nature of the data

allows for temporal ordering of exposure and outcomes thus strengthening causal inference where significant associations are observed. This would not be possible with cross-sectional data as these are not collected on the same individuals over time. Since demographic surveillance allows for collecting different pieces of social and health information on individuals and households, these data provide an opportunity to explore recent changes in adult mortality in this population and allows us to relate variables measured along the way to the outcomes. Measurement of adult mortality is direct and thus no assumptions about the population are made as is often the case with the indirect methods of estimating all-cause adult mortality.

Although this study has several strengths, it also has some limitations. Health and demographic surveillance is inevitably restricted to a relatively small population due to financial and logistical limitations. The small population under surveillance makes it hard to generalise results from the surveillance to the national population in the way that one is able to do with national survey data. Because they involve repeated data collection waves several times a year for many years and collect a large volume of data, surveillance systems are expensive to manage and not all governments that need these data are willing to fund such undertakings. Another challenge is that continued surveillance may result into respondent fatigue and this might affect their participation and as such the quality of data may worsen with time. The verbal autopsy tool, while useful in identifying probable causes of death in majority of cases, has limitations in ascertaining causes of death that present with ambiguous symptoms. Because of this, the relative contribution of the various causes of death to the overall mortality burden cannot be known with certainty. Close to 30% of all deaths in this study did not have cause-of-death data. However, further analyses using multiple imputation methods showed that known characteristics of people who died from unknown causes did not differ greatly from those of people who died from known causes and as such estimates from observed and imputed data were very similar.

Support from other types of data such as HIV surveillance or the validation of the verbal autopsy tool would have gone a long way to increase confidence in the cause of death data used here. For example, it would be useful to see what proportion of those diagnosed as having died of HIV/AIDS, as per verbal autopsy, also have a positive HIV

test. Unfortunately, validation has not been undertaken yet and no linkable HIV serology data are available for this population.

As has been demonstrated in chapter 6 and other earlier studies, migration is quite intense in this population <sup>[100, 133]</sup>. Migration may result into loss to follow up in the surveillance system. Migration and eventual loss to follow up may be related in some way to poor health and imminent death, as reported in pre-anti-retroviral therapy (ART) era studies in South Africa and Kenya <sup>[105, 367]</sup>. If ill-health related migration occurs, then mortality in the source population can be severely underestimated while that for the receiving population might be overestimated due to deaths of recent returnees who were terminally ill. With increasing access to ART, this is likely to become less common as this treatment is more accessible in urban than in rural areas, which are the preferred destination for terminally-ill adults. However, as ART is only partially effective at keeping people with AIDS alive, this probably would not completely reverse the stream of ill-health related migration to rural areas.

### Conclusions, policy and research recommendations:

HIV/AIDS is a leading cause of adult death, particularly among women. Efforts to prevent and treat HIV/AIDS should be scaled up to reach all adults in the informal settlements. In this population voluntary counselling and testing among females aged 15-49 years is about 33% while that for males 15-54 years is about 29%. Provider initiated counselling and testing is about 29% among women and only 9% among men and close to 50% of adults have never tested for HIV before <sup>[368]</sup>. As an entry point into care and treatment, voluntary counselling and testing and provider-initiated counselling and testing should be promoted to reach all adults, including those who are not sick and those seeking care for any illness respectively.

While tuberculosis is curable, it remains a major cause of death among adults in this population <sup>[1]</sup>. The success of tuberculosis prevention and control lies in early case detection and provision of effective treatment with emphasis on adherence to the treatment regime. Many tuberculosis cases remain undiagnosed in Kenya <sup>[94]</sup>. Control of tuberculosis is important as tuberculosis complicates HIV/AIDS and vice versa resulting in higher rates of treatment default, treatment failure and development of drug



resistance. The two disease entities contribute enormously to the overall burden of adult mortality and therefore, control efforts, including case detection and treatment, should be scaled up coupled with effective monitoring of mortality and other health outcomes attributable to tuberculosis.

Injuries, mainly among men, are a leading cause of death. Unintentional injuries, for example, road traffic crashes, falls and burns are common, as are intentional injuries such as firearm injuries, lynching and other forms of interpersonal violence. The poor presence of law enforcement, high crime rates and lack of emergency services all contribute to the huge burden of deaths from injuries. Kenya does not have a comprehensive injury prevention policy to guide implementation of prevention activities. This is urgently needed to reduce the carnage from injuries. So far, the efforts have been less than comprehensive. For example, with the recognition that firearms are a major cause of injuries, the government of Kenya has come up with a National Action Plan to help control small arms. Also, recently there have been revisions to the traffic panel code to provide for heavier penalties for traffic rules offenders. However, other major causes of injuries have so far not been addressed. For example, there is no effective enforcement of safety measures in households and workplaces. Emergency and rescue services in case of accidents and fires also remain very limited. Creating public awareness and developing an injury prevention policy would be a good starting point from which to address this issue. Policing should be more responsive to community security needs and probably community policing should be increased to avert potential crime and ultimately ensure that residents are more secure.

Future research in this population and Kenya in general should focus on expanding surveillance of the top causes of death to a larger population. In the absence of vital registration in many sub-Saharan African countries, donors and governments should support researchers in maintaining and/or establishing more surveillance systems as a first step toward generating nationally representative vital statistics in the country. This will not only help estimate levels and trends of adult mortality and other statistics, it will also help provide data for monitoring and evaluation of disease-specific program performance for example treatment outcomes for HIV/AIDS or tuberculosis. Also, while the verbal autopsy tool from which the tool used in the NUHDSS was adapted has

been validated in several places, there is need for local validation to increase confidence in the cause-of-death data.

### **12.3 Impact of adult deaths on children: Revisiting the conceptual framework**

The next part of the discussion focuses on the impact of adult death on child well-being. First, I revisit the conceptual framework which partly guided the analysis to appraise to what extent the findings have changed or confirmed the conceptual framework as proposed before the analysis (Figure 23). Where relationships were either confirmed or modified, the text is italicised and presented in blue for the predictor variables or red for the outcome variables to highlight and emphasise what has been discovered. Some of the variables in the initial conceptual framework diagram have been broken down further to highlight the actual relationships between predictors and outcomes in this population. An example is parental death. This has been broken down to maternal and paternal deaths.

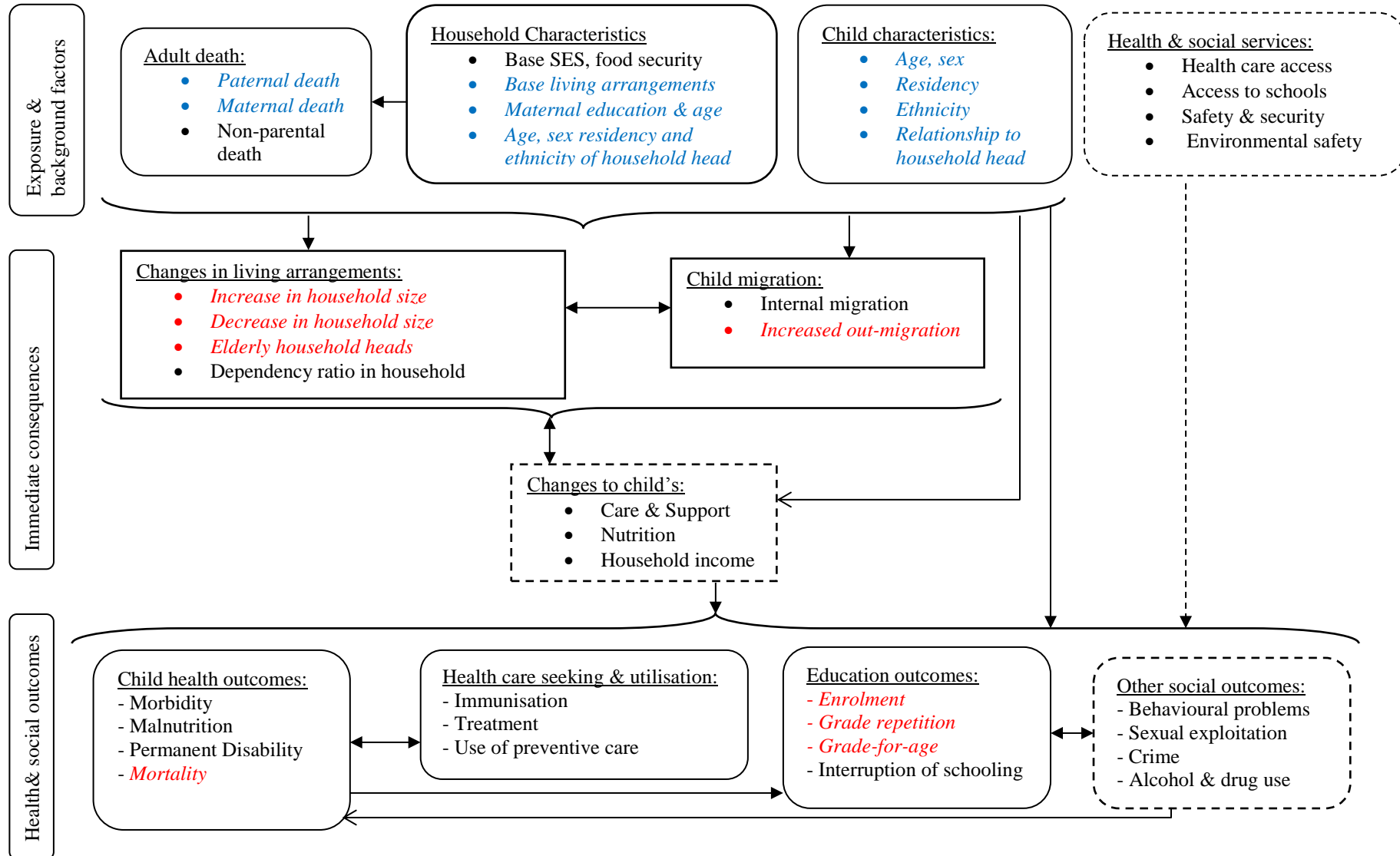
I use examples from the findings to illustrate how the conceptual framework has changed or been confirmed. The results showed that not all adult deaths have the same impact on child well-being. Non-parental deaths did not have a significant impact on either the social well-being or health of children and therefore the distinction between parental and non-parental death is a very important one to make when assessing impact of adult death on children. Furthermore, even for parental deaths, the impact on children often depends on which parent dies. The impact may also be in opposite directions depending on the health and social outcomes under consideration.

While paternal death resulted in an increase in average household size, maternal death resulted in a decrease in average household size. On the other hand, paternal deaths were associated with households being subsequently headed by an elderly person, while maternal deaths were associated with increased risk of child outmigration. Maternal deaths, but not paternal death, were associated with a significantly higher risk of child death. Also, maternal deaths were associated with poorer educational outcomes for the residents of Viwandani. Generally, adult deaths were not significantly associated with immunisation outcomes. Children's socio-demographic characteristics, especially age

and ethnicity and some of the household-level factors were associated with child outcomes as has been documented in other studies.

Overall, the key results are in agreement with the conceptualisation in the framework but the study has identified specific relationships as opposed to the generalisations outlined in the initial conceptual framework. A detailed discussion, recommendations and policy implications for each of the outcomes are given below.

Figure 23: Revised Conceptual Framework



## **12.4 Impact of adult deaths on children: migration and living arrangements**

The second aim of the thesis was to estimate the impact of adult death on children's migration and living arrangements. Like adults, children in Korogocho and Viwandani slums exhibit high levels of migration within and out of the slums. Child migration is more intense in Viwandani slum than Korogocho slum. The findings showed that child migration and changes in their living arrangements subsequent to adult death depend on how the children are related to the deceased adult.

Death of a mother is generally associated with higher odds of child out-migration from the slums while death of other adults in household is not significantly associated with child out-migration. Net of other factors, the risk of child out-migration decreased with increasing household size. Irrespective of their exposure status, children living in smaller households had a higher tendency to out-migrate from the slums, implying that larger households probably draw on their membership to provide care to children or generate income to sustain them. Death of an adult takes away a potentially productive household member. This may be a particular challenge for small households, which may have far too few hands to contribute to income generation and child care after an adult death. Ultimately, this shortfall might be the incentive for moving children out to destinations where they can be cared for.

Children who live in households that experience an adult death are less likely to move to other households within the slums. However, on average, more children change place of residence before death occur in a household, implying that to some extent child placement is planned prior to adult death. As such, death may not be seen to be associated with the risk of children migrating within the slums. Death of a father is associated with an increase in household size, on average, while death of a mother or other adult is associated with a decrease in household size. In sub-Saharan Africa, when a mother dies, children tend not to live with their surviving father while, if a father dies, children usually live with their surviving mother <sup>[16, 291]</sup>. Because widowed mothers tend to live with their children, they seem to recruit other individuals to their households to help with child care and/or income generation, hence increasing their household's size. Maternal orphans tend to be fostered by relatives who can provide child care, such as

grandparents or aunts, even when their father is alive. Additionally, the decrease in household size observed when a mother dies might be partly due to the higher likelihood of her young children dying, further reducing the household's size [165, 168, 173].

Children living in households that experienced an adult death are more likely to live in households with an older household head than those who lived in unaffected households. The effect of an adult death in a household on subsequent household headship is graver in Viwandani slum than Korogocho. Because Viwandani households are generally smaller than those in Korogocho, death of one adult, for example in a single-parent household, leaves the household with no other adult. This exposes the household to the risk of being headed by a child or elderly individual, who may not have adequate means to fend for the household. The potential consequences of this may include some household members migrating out to other destinations while other members stay. Alternatively, the entire household may dissolve with members out-migrating or joining other households, leaving none of the members in the original household.

Other than death of an adult in household, there are other independent determinants of child migration and changes to their living arrangements. Contrary to expectation, children living in households headed by a non-prime age household heads are less likely to out-migrate or move within the slum. It is likely that such children are orphaned in the first place, and thus may not have any destinations to which they can migrate. Children in male-headed households are more likely to out-migrate and this may indicate that lack of child care is a key motivation for child migration, for example when a mother dies.

While adverse situations like bereavement may push children to out-migrate, children with better socio-economic backgrounds (higher wealth index, higher maternal education) also have a higher tendency to out-migrate, probably for reasons such as schooling or to move to a better neighbourhood. The fact that households with non-parent heads tend to be bigger than those where the household head is the parent to the child suggests that such households are often recipients of children from elsewhere

because of their relatively better socio-economic status and/or the presence of adults who can provide child care. Households with older children (5 years and above) are generally larger than those with children less than 5 years of age. Larger households are more likely to be headed by non-parent heads, or older individuals. Lower maternal education and residency in Korogocho slum were also associated with larger households.

### Strengths and limitations:

Continuous surveillance allows monitoring of the population over time and recording of events as they occur. Importantly too, each individual can be monitored as they move in, out of, or between slums, but also from one household to another. Each household has a household head whose relationship to everyone else in the household is known and thus relationships between all household members can be constructed. Individuals who ever lived in the surveillance area retain their personal identification even when they migrate out and come back or change household and thus they can be followed up over time without losing or mixing up their identity.

The frequent update of residents' statuses in the surveillance system allows for capturing of relatively short-stay migrants and changes in living arrangements. This is very important for a population that exhibits circular migration with variable periods of absence and stay in the slums <sup>[100]</sup>. In the NUHDSS, migration is defined by duration of stay in or out of the slums not intent as is the case with other surveillance sites. Using intention to stay or not stay to define a migrant was avoided as intentions can change anytime for various reasons and, as such, a lot of false residents could be generated. Of course the 120-day cut off is arbitrary, but it allows for weeding out of short-stay visitors and those whose intent to live in the slums changed shortly after their arrival.

Attrition from the surveillance area is a major challenge. Individuals who out-migrate may never come back to the surveillance area and therefore their social and health outcomes after migration are never established to fully understand the consequences of out-migration. This means that the surveillance data may underestimate the adverse impact that adult deaths have on children's well-being. Selective out-migration of children who lose a mother may result into biased estimates between orphans and non-

orphans as the potentially more disadvantaged maternal orphans are systematically lost and their outcomes not known.

A substantial number of children under surveillance cannot be linked to their parents because either their parents do not live in the surveillance area or they live in the surveillance area but in different households from the children. Currently there is no mechanism for identifying and linking parents to children living in different households. The surveillance system does not also ascertain the survival status of non-resident parents and thus this is not accounted for. The impact of orphanhood on those children who are orphaned but whose orphanhood status is unknown cannot be measured and this may lead to misleading conclusions about the overall consequences of orphanhood for children.

### Conclusions and research recommendations:

Death of adults in a household, particularly parents, impacts on children's migration and living arrangements. Children who lose a mother tend to out-migrate from the slums, live in smaller households and in households with older heads. On the other hand, paternal orphans do not have increased risk of out-migration, but tend to live in larger and poorer households. These dynamics have social and health ramifications for the affected children with regard to their survival, health-care seeking and education as examined in chapters 8 to 11. The literature on migration has shown mixed impacts on migrants, including children, and this has to be kept in mind when considering migration as an intermediate variable in the pathway between adult mortality and child health and social outcomes <sup>[111, 122]</sup>.

In the future, attempts need to be made to understand the motivation for migration and consequences of migration, at least in its immediate aftermath. Some individuals lost to follow-up might remain within the slums but invisible to the surveillance system and yet end up being misclassified as having out-migrated. This is thought to be due to deliberate falsification of identity (name). Anecdotally, this has been particularly associated with young men with criminal backgrounds who wish to conceal their true identity. Additionally, continuous surveillance may result into community fatigue due to repeated rounds of interviews which bring no visible individual material benefit. While



following up of out-migrants is quite expensive, as they go to diverse destinations, it might be worthwhile to invest in a one-off study to try and understand what happens to them after migration. This is important because, while migration can potentially interrupt schooling or access to health care, it does not always follow that migration has negative effects. Instead, a move may provide the child with better opportunities than does continued residence in the slums.

## **12.5 Impact of adult deaths: child survival, immunisation and education**

The third aim of the thesis is to estimate the impact of adult deaths in households on children's survival, health care utilisation (immunisation) and educational attainment. Infant and under-five mortality rate in the two slums is high. The neonatal mortality rate was 14 deaths per 1000 live births, infant mortality was 50 deaths per 1000 live births and under-five mortality rate was 78 deaths per 1,000 live births.

Pneumonia, neonatal causes, diarrhoea, measles, malnutrition and HIV/AIDS/TB are the leading causes of death for children aged less than 5 years. Malaria was not among the top causes of under-five death in this population. Neonatal causes of death include pneumonia, prematurity, birth asphyxia, septicaemia and diarrhoea. The high contribution of asphyxia (intrapartum causes) and septicaemia to neonatal deaths points to poor obstetric and newborn care in the slums and probably poor maternity care seeking behaviour among mothers in the slums <sup>[96, 327]</sup>. Full immunisation coverage for children 12-23 months is about 78%, comparable to that of Nairobi province at 73%. Although the coverage for the slums seems impressive, it is well below optimal coverage. Given the crowding and therefore close personal contact in slums, a higher level of coverage might be needed to achieve herd immunity than in less congested areas <sup>[189]</sup>.

Death of a mother, but not that of other adults in the household, increases the risk of child death by about 3 times compared with children living in households with no adult death. This finding is in line with similar findings in other studies <sup>[6, 165, 173]</sup>. The risk of child death is highest in the 6 months following death of a mother but is also substantial in the 6 months preceding the death of a mother. Studies that only look at orphans are

incapable of estimating impact due to terminal illness. Indeed, very few interventions target children of terminally-ill guardians. In situations of protracted terminal illnesses, as is often the case with HIV/AIDS, children of terminally-ill guardians may be the most vulnerable of all children. While orphans are often absorbed by the extended family system, children whose parents are terminally ill may continue living with their ailing guardians enduring the effects of reduced earnings to the household, poor nutrition, reduced care and support. Older children may miss school to provide nursing care or go to work to generate income to support their parents. Additionally, the stigma and discrimination towards AIDS patients may be extended to their household members resulting into emotional distress for the children.

Death of an adult in a household is associated with a significantly higher risk of child death from a number of causes, including pneumonia, diarrhoea, HIV/AIDS/TB and neonatal causes. Insufficient evidence exists to conclude that death of an adult in a household increases the risk of a child not being fully vaccinated or delayed receipt of the respective vaccines. This might be due to the fact that childhood immunisation is offered free-of-charge with the only cost involved being that of transport to a vaccination centre and the time spent. With relatively high immunisation coverage, any differences by other factors are likely to be minimal, difficult to tease out and probably may not require any intervention.

As expected, the risk of child death decreased with age with the sharpest decline being after the neonatal period. As with adults, mortality was highest among the Luo and Luhya ethnic groups. Among adults, HIV/AIDS is the main driver for the high mortality in these ethnic groups. Children whose mothers or fathers died of HIV/AIDS/TB were more likely to die of the same illness than other children. Additionally, those who do not die from HIV/AIDS suffer the effects of orphanhood attributable to HIV/AIDS after death of their parents.

Surprisingly, maternal education was not significantly associated with child survival but was positively associated with children's full immunisation status. Children who were born to teenage mothers had a higher risk of death than those whose mothers were aged between 20 and 39 years of age at the time the child was born. For all children less than

2 years of age, slum of residence, ethnicity, maternal education and age were associated with delay in receiving vaccines.

Viwandani children who lost a mother generally have poorer educational attainment than other children in the same slum. They were less likely to be enrolled in school by age 7, more likely to repeat grades, more likely to interrupt schooling (not significant) and more likely to be of an inappropriate grade-for-age. For children living in Korogocho, death of a mother was not associated with significantly poorer educational attainment. Indeed, for some outcomes such as grade repetition and interruption of schooling, the association was in the reverse direction to that in Viwandani in spite of the fact that Viwandani slum children have better educational attainment, net of other factors, than those in Korogocho.

Other independent predictors of schooling outcomes include gender, ethnicity and maternal education. Boys were less likely to be enrolled in school by age 7 and they were more likely to be in an inappropriate grade for their age than girls, while there is no significant gender difference in risk of interruption of schooling or repeating a grade. Children of Kikuyu ethnicity had better schooling outcomes than the other children with the exception of grade repetition and interruption of schooling where there were no significant differences. The children whose mothers had secondary or higher education were more likely to be enrolled in school by age 7 and less likely to be in an inappropriate grade-for-age.

Previously, there has been great concern with regard to the schooling of girls. However, as other recent evidence shows, Kenya has already achieved gender parity in primary education. It has been estimated that by end of year 2010, 83% and 82% of girls and boys were enrolled in primary schools respectively, giving a gender parity index of 1.01 in favour of girls <sup>[369]</sup>. It might be the right time for the attention to shift to examining possible schooling disadvantages at secondary education level where the gender parity index is estimated to be 0.94 in favour of boys. The observed differences in schooling outcomes by ethnicity and maternal education may point to differences in household resources that facilitate schooling such as finances, emotional support, housing and guardians' interest, which have not been directly measured in this study.

### Strength and limitations:

The surveillance data used here allow for linking of adults and their survival to children's social and health outcomes over time as the relational database identifies individuals and the households they belong to over the period of surveillance. Cause-of-death data for both adults and children are available and this allows for examination of the relative importance of the different causes of adult death for various child outcomes. It also provides an idea of the likely causes of death among orphans and vulnerable children. For example, from the results, it is clear that children whose parents die of HIV/AIDS are themselves likely to die of HIV/AIDS, indicating that vertical transmission of HIV is a major issue in this population.

The education status of all school-going children is updated every year and this allows for measurement of school participation with a fair level of accuracy. This study examined more than one schooling outcome and this contrasts with several previous studies that have looked only at one or two indices of educational attainment. The analytical approach and methodology were robust and took care of repeated events and potential clustering within the individual.

Attrition is a potential problem with regard to the estimation of child mortality and other outcomes. The results here showed that exposed children were more likely to die but also that they were more likely to migrate out of the surveillance area. This means that the count of deaths is likely to be underestimated. The scale of this problem cannot be quantified as out-migrants are not routinely traced outside of the surveillance area. As a consequence, loss to follow-up might lead to underestimation of child mortality and other outcomes that might be associated with migration. Also, because parental survivorship for children who are born to parents who live outside of the surveillance area or who do not live with their children is unknown, the impact of the death of such parents is not captured and is potentially underestimated for the population under study.

Ascertaining the cause of a child's death using a verbal autopsy is often difficult. About 40% of all neonatal deaths with an unknown cause had ambiguous symptoms.

The verbal autopsy tool has low sensitivity and specificity for certain childhood illnesses such as malaria and neonatal causes of death <sup>[267, 324, 328]</sup>. This means that a sizeable proportion of child deaths are misclassified and this might have undesirable programmatic effects as the wrong priorities may be identified.

A sizeable proportion of the immunisation data was obtained through recall. Respondents may give socially desirable answers to field workers regarding child care and might misrepresent the immunisation status of their children. Secondly, it is possible that assessment of delay in this study is biased as the children whose immunisation status reports were based on recall were excluded from assessment of delay. This could potentially result in selection bias especially if the children with no immunisation record are likely to be those whose guardians are less concerned about the children's well-being and tend to have children immunised late or opportunistically when they go to a health facility for other medical reasons.

The data on schooling do not capture short spells of school interruption and yet these may have cumulative effects resulting into poor progress or eventual drop out of school. Also, the data do not include an assessment of individual children's performance at school. Even if children attend school, challenges in their households such as bereavement may affect their concentration resulting in poor grades, leading to low motivation and eventual dropout of school.

The educational attainment outcomes analysis contained in this thesis is restricted to children between 6 and 14 years. By convention childhood ends at 17 years. However, I focused on primary school participation, which is targeted by the millennium goal on education. Additionally, adult deaths were defined as deaths occurring to individuals aged 15 or more, in line with the conventional demographic definition of adult mortality and therefore these two categories were kept mutually exclusive.

Lastly, not all determinants of school participation such as quality of schools were controlled for in the analysis. This may bias the estimates. Sudden and short-term changes in household income may happen after parental death and this may subsequently impact on children's school participation. However, I lacked an

appropriate measure of changes in socio-economic status capable of capturing such changes. The household wealth index constructed from household asset ownership does not capture short-term changes in household finances.

### Conclusions, policy and research recommendations:

Maternal orphanhood negatively impacts child survival and education but has little or no impact on child immunisation. These findings are largely in line with expectation given the crucial role that mothers play in child care, support and nutrition. Children's living arrangements after a maternal death seems to mediate the negative impact of such deaths on children's health and education. While the residents of Viwandani slum are on average wealthier and have better child survival and schooling outcomes than those in Korogocho, their households are typically smaller. Death of an adult in a small household leaves children in an awkward situation with limited support and thus at greater risk of migration or living in unfavourable arrangements that subsequently reduce their survival and schooling opportunities.

Most of leading causes of child deaths have cost-effective interventions and therefore many child deaths could be averted if these interventions were made available and utilised in this community. Every woman should deliver with the assistance of a professional care provider to stem the big burden of neonatal deaths that are attributable to poor obstetric care. Newborn care also needs to be improved to reduce neonatal deaths. Paediatric HIV/AIDS deaths need to be controlled by ensuring that all pregnant women know their HIV status and that those who are found to be HIV positive are given anti-retroviral drugs to prevent transmission to babies.

Research examining why children from certain ethnic backgrounds have a higher risk of death from diarrhoea, measles and malnutrition than others with whom they live in the same environment should be considered. Designing of health messages should try to target sub-groups identified here as being more vulnerable. For example, among the Luo, many of the children diagnosed to have died of either HIV/AIDS or malnutrition by the verbal autopsy are often thought by their families to have died of *chira*- a curse associated with breaking taboos <sup>[370]</sup>.

Turning to the surveillance system, efforts must be made to reduce the proportion of deaths with undetermined cause of death. Two issues need to be addressed here: i) insufficient information that does not allow for arriving at a cause of death, and ii) failure to conduct a verbal autopsy. Insufficient information could be related to interviewer skills on eliciting responses but it could also be related to getting information from inappropriate respondents who lack detailed knowledge of the deceased's illness. There might be need for re-training of and provide feedback to data collectors after each wave of data collection. The need to interview credible respondents should be re-emphasised in order to collect reliable and sufficient data. At the level of assigning cause of death, physicians may arrive at different codes and fail to agree and as a result classify the cause of death as unknown. The physicians should attempt as much as possible to agree on a cause and only assign "unknown" as a last resort, not a convenient one whenever there is disagreement. With recent attempts to automate assignment of cause of death using algorithms, validation studies should be carried out to test their performance and use. This could help improve the coding process and also improve reproducibility and comparability of verbal autopsy data across surveillance sites. Physicians who assign cause of death also need to be oriented on coding, paying attention to the potential public health utility of the data being collected. The challenge of verbal autopsies not being conducted could be addressed by devising new ways of early reporting of deaths to reduce the time lag between death and interview as the risk of out-migration and recall bias increases with time. Generally, verbal autopsy interviews should be conducted soon after death instead of waiting for the routine data collection waves. Since most deaths are reported to the area chief as a legal requirement, working with the chief's office to identify recent deaths could be explored.

Should maternal orphans in Viwandani be targeted? Some authors propose targeting of orphans where negative impact has been demonstrated, while others support reducing socio-economic inequalities in communities in general <sup>[23, 352]</sup>. While the larger households found in Korogocho might be important in keeping affected children in school, it does not make the situation of the children in this slum much better. Korogocho households are poorer and on average experience more adult deaths and have worse health and education indicators. Therefore based on this, targeting Viwandani maternal orphans might not be helpful. In this population studied, it appears

that poor schooling outcomes are more related to the degree of care and support provided at a household level than lack of financial support. A more holistic approach to improving slum education might be a better approach than targeting only orphans in particular slums. However, while I would argue that targeting Viwandani orphans might not be very useful, it should not be forgotten that the “family” is important in keeping children alive and in school after death of their guardians. A potential intervention here is to encourage guardians whose death is predictable and imminent to make decisions about how their children will be fostered by relatives or willing community members. A similar intervention involving succession planning and identifying standby guardians prior to parental death was tried in Uganda and the evaluation conducted of it suggests that it was very helpful <sup>[371]</sup>.

Lastly, more research relating orphanhood and individual children’s school performance is needed with better controls for household socio-economic status. Also more research, probably anthropological in design, should examine and put into context the schooling disadvantage of maternal orphans in Viwandani.

## **12.6 Concluding remarks**

The findings discussed in this thesis confirm that children who have lost an adult in their households are more vulnerable than other children. This has been demonstrated for children’s living circumstances and their health and educational outcomes. Children’s vulnerability to increased mortality begins while their parents are ailing and continues thereafter. Maternal orphans in particular are at an increased risk of death. They also tend to live in larger and poorer households, mainly headed by older household heads and have poorer education outcomes, more so in Viwandani slum and when they come from smaller households. While this study examined a limited range of child well-being measures, these give an indication of how adult deaths in households impact the well-being of children in slums generally. However, due to data limitations, it not clear how and to what extent changes in household economic fortunes and/or lack of care and support mediate and explain the negative impact of adult death on children. Exploring this would require more detailed data and possibly a detailed assessment of child care and decision making at household level.



The findings here should be viewed in the context of the pre-existing vulnerability of children by virtue of this population living in a slum environment. The majority of children in the slums live in extreme poverty, insecure neighbourhoods, poor housing and a generally unhygienic environment. They also have limited access to health care facilities and formal schools, they are highly mobile and have early exposure to social ills- including unsafe sex, alcohol, drugs and gang culture <sup>[37, 357, 358]</sup>. The recent increase in adult mortality- mainly from HIV/AIDS and corresponding increase in the number of orphans compound the situation in the slums further. In sub-Saharan Africa, the majority of orphans are cared for by the extended family <sup>[372, 373]</sup>. However, with the ever growing number of orphans and a limited available pool of relatives to take care of them, some orphaned and vulnerable children end up living in unfavourable arrangements. For example, some OVC live in households with a child or elderly person as the household head, some live on streets, while others live with non-relatives <sup>[19, 145, 146]</sup>. This is happening against a background of shrinking extended families, and increases in the proportions of lone-parent households and non-resident parents. Typically the average household size in the slums is less than 3 people and this means there are very few adults to provide child care and or contribute to income generation, especially after adult death <sup>[133]</sup>. This is particularly of concern as most working adults in this population do not have formal social protection, such as life insurance, which would help mitigate the financial losses occasioned by an adult death. In the absence of formal safety nets and shrinking traditional extended family support, death of a bread winner may spell catastrophe for their dependants. The household and community resources available in rural areas are not readily available in urban areas. Slums are “a cash economy” with most residents looking for paid work to sustain themselves and their families and having little time for child care. While most rural households grow their own food and can draw on the extended family for support, slum residents must earn cash to be able to buy all they need and at the same time have limited external support to draw on.

As the results showed, children’s vulnerability may rise even before death of their guardians. Vulnerability may emanate from loss of household income due to terminal illness, diminished parental care and support and as a result some children may withdraw from school to help care for the sick or work to generate income. In the case

of HIV/AIDS, children of infected parents have a higher risk of being infected themselves and they are more likely to be stigmatized and discriminated against by community members <sup>[374, 375]</sup>. Following parental death, some of the stresses endured during the terminal illness may be accentuated through funeral costs, emotional distress, and the separation of siblings, dropping out of school, ill-health, child labour and sexual exploitation with associated risk of HIV infection.

Moving forward, there is need to implement interventions to reduce adult mortality, particularly that attributable to HIV/AIDS and injuries, and the same should be done for children. Targeting maternal orphans in slums through family-centred support could also be considered but pervasive poverty even among non-orphans might defeat the purpose. While material support is important for child health and education, the findings here imply that non-material household resources such as child care and support are crucial in mitigating children's vulnerability. Programming aimed at supporting orphaned and vulnerable children normally emphasise supporting them within a family environment <sup>[33, 223]</sup>. However, in the slums, due to the limited nature of the extended family, some OVC may not be taken in by relatives and probably end up on the streets. For this reason, OVC programming in slums might consider supporting institutionalised OVC care side-by-side with family-centred support. A few volunteer operated "OVC homes" already operate in the two slums. Unfortunately, they have serious financial constraints as they cannot compete for grants with mainstream implementers who conform to the funders' strategy of providing family-centred support.

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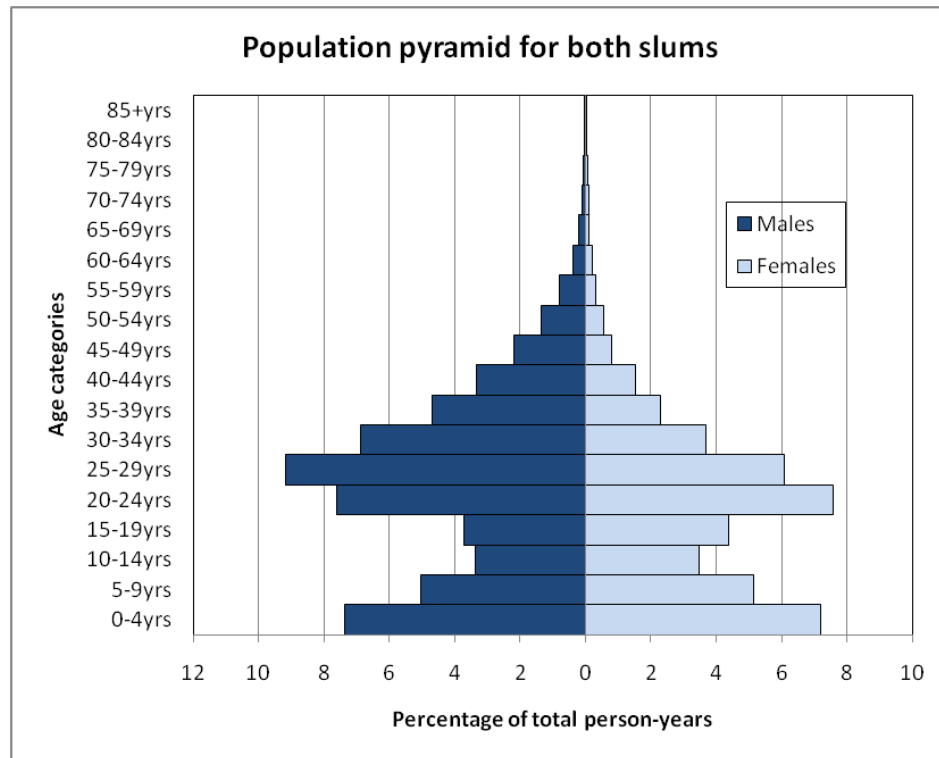


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## Appendices:

Appendix I: Population pyramid for both Korogocho and Viwandani slums in Nairobi city



## Appendix II: Comparison of life expectancy among adults at Nairobi and other demographic surveillance system sites in sub-Saharan Africa

Age group	Nairobi DSS (2003-2007)	Hai DSS (1994-1999)	Agincourt DSS (1995-1999)	Niakhar DSS (1995-1998)	Gwembe DSS (1991-1995)	Farafenni DSS (1995-1999)	Bandim DSS (1995-1997)	Butajira DSS (1995-1997)	Manhica DSS (1998-1999)	Oubritenga DSS (1995-1998)	Navrongo DSS (1995-1999)
<b>Females</b>											
15-19yrs	41.4	54.3	62.4	54.0	52.4	53.6	37.4	52.4	51.9	54.1	48.3
20-24yrs	36.9	49.7	57.7	49.8	47.9	49.4	32.9	48.3	47.6	50.0	43.9
25-29yrs	32.9	45.9	53.3	45.7	44.2	45.5	29.1	44.1	44.1	46.0	39.7
30-34yrs	29.6	42.7	48.9	41.6	40.7	41.4	25.6	40.1	40.7	42.1	35.8
35-39yrs	27.6	39.9	44.7	37.2	37.6	37.7	22.3	36.4	37.1	38.2	32.1
40-44yrs	25.4	36.8	40.6	32.8	34.2	33.6	18.8	32.3	32.9	34.2	28.0
45-49yrs	23.0	33.5	36.6	28.8	30.6	29.4	16.3	28.4	28.9	30.4	24.0
50-54yrs	20.5	29.9	32.3	25.1	26.8	25.4	13.8	25.5	25.5	26.3	20.2
55-59yrs	18.7	26.0	28.2	21.2	24.6	21.4	11.5	21.5	22.0	22.5	16.8
60-64yrs	16.4	22.3	24.0	17.6	20.5	18.0	10.4	18.8	19.7	18.6	13.8
65-69yrs	14.3	18.5	20.7	14.0	16.6	15.1	9.6	16.1	16.9	14.9	11.1
70-74yrs	11.7	15.1	17.5	10.9	13.5	12.6	6.9	13.8	13.7	11.8	9.1
75-79yrs	12.1	11.7	15.1	8.3	12.2	10.8	5.7	12.1	11.9	9.5	7.9
80-84yrs	9.0	8.6	12.9	6.1	8.6	9.7	4.6	10.6	10.2	7.6	7.0
85+yrs	10.1	5.6	12.0	4.3	8.0	9.7	4.6	10.2	9.2	5.6	6.6
<b>Males</b>											
15-19yrs	43.9	48.1	53.7	50.6	47.1	48.6	34.8	51.0	41.7	50.3	44.4
20-24yrs	40.1	43.7	48.9	46.3	42.7	44.1	30.6	47.0	37.4	45.9	40.0
25-29yrs	36.2	39.6	44.4	42.1	38.1	40.0	26.6	43.3	32.9	41.5	35.7
30-34yrs	32.3	36.0	40.3	38.2	34.5	35.3	23.2	39.7	29.4	37.8	31.8
35-39yrs	29.0	33.1	36.4	33.8	32.4	31.3	19.8	35.6	27.5	34.4	28.3
40-44yrs	25.8	30.5	32.9	30.1	30.6	27.6	16.2	31.8	24.8	30.9	24.8
45-49yrs	23.3	27.8	29.1	26.1	28.1	24.4	13.9	28.6	22.8	27.2	22.0
50-54yrs	20.2	25.1	26.2	22.1	24.5	20.7	12.3	25.7	19.8	23.6	19.1
55-59yrs	17.5	22.3	23.0	18.9	21.9	18.0	9.7	22.2	17.4	20.0	16.4
60-64yrs	14.9	18.9	20.2	15.3	19.7	14.7	7.7	18.7	15.8	16.7	14.0
65-69yrs	11.7	15.8	17.1	12.6	18.0	12.3	6.3	16.4	13.6	13.2	12.0
70-74yrs	10.5	12.9	14.7	10.2	13.6	10.5	5.9	13.7	11.3	10.7	10.3
75-79yrs	9.7	10.3	12.7	8.0	10.7	8.6	5.5	12.5	9.1	8.3	8.3
80-84yrs	8.7	7.9	11.9	6.0	9.0	7.2	5.4	9.9	7.1	6.4	7.6
85+yrs	7.4	5.4	10.8	3.9	4.0	7.1	5.0	7.1	5.0	5.1	6.3

DSS: Demographic Surveillance System. \*Comparison data were published in "Population and Health in Developing countries" [55]

### Appendix III: Regression result from a Cox proportional hazard model and piecewise exponential model for comparison

Variables	Results from a Cox Model			Results from a piecewise exponential model		
	Hazard Ratio	(95% CI)	P value	Hazard Ratio	(95% CI)	P value
15-19yrs				1.00		
20-24yrs				1.52	(1.14;2.05)	0.005
25-29yrs				2.49	(1.88;3.30)	<0.001
30-34yrs				4.44	(3.37;5.85)	<0.001
35-39yrs				4.59	(3.45;6.11)	<0.001
40-44yrs				5.54	(4.15;7.40)	<0.001
45-49yrs				5.19	(3.79;7.12)	<0.001
50-54yrs				6.79	(4.91;9.38)	<0.001
55-59yrs				7.18	(5.01;10.27)	<0.001
60-64yrs				7.07	(4.63;10.81)	<0.001
65-69yrs				11.78	(7.55;18.36)	<0.001
70-74yrs				16.80	(10.73;26.32)	<0.001
75-79yrs				11.93	(6.59;21.60)	<0.001
80-84yrs				20.10	(11.56;34.95)	<0.001
85+yrs				38.04	(23.71;61.04)	<0.001
Gender						
Females	1.00			1.00		
Males	0.87	(0.78;0.97)	0.009	0.87	(0.78;0.97)	0.011
Slum						
Korogocho	1.00			1.00		
Viwandani	0.73	(0.61;0.87)	<0.001	0.74	(0.62;.88)	0.001
Ethnicity						
Kikuyu	1.00			1.00		
Luhya	0.82	(0.69;0.97)	0.024	0.82	(0.69;0.97)	0.023
Luo	1.75	(1.53;2.01)	<0.001	1.75	(1.53;2.01)	<0.001
Kamba	0.59	(0.50;0.70)	<0.001	0.59	(0.50;0.69)	<0.001
Meru	0.74	(0.43;1.28)	0.282	0.74	(0.43;1.28)	0.279
Embu	0.62	(0.29;1.31)	0.212	0.62	(0.29;1.30)	0.207
Kisii	0.46	(0.30;0.71)	<0.001	0.46	(0.30;0.70)	<0.001
Somali	0.42	(0.26;0.70)	0.001	0.45	(0.28;0.74)	0.002
Garre	0.55	(0.36;0.86)	0.008	0.53	(0.34;0.82)	0.005
Borana	0.86	(0.59;1.24)	0.413	0.87	(0.60;1.26)	0.475
Other	0.56	(0.37;0.84)	0.006	0.56	(0.37;0.84)	0.005
Wealth						
Poorest	1.00			1.00		
Middle	0.88	(0.75;1.05)	0.154	0.89	(0.75;1.05)	0.175
Wealthiest	0.83	(0.73;0.95)	0.007	0.84	(0.73;0.96)	0.010
Unknown	1.95	(1.35;2.80)	<0.001	1.96	(1.36;2.82)	<0.001
Education						
No education	1.00			1.00		
Primary	0.88	(0.72;1.08)	0.215	0.85	(0.70;1.03)	0.092
Secondary & higher	0.66	(0.53;0.83)	<0.001	0.64	(0.51;0.79)	<0.001
Unknown	0.88	(0.62;1.24)	0.473	0.84	(0.60;1.18)	0.319
Calendar year						
2003	1.00			1.00		
2004	0.88	(0.75;1.02)	0.091	0.87	(0.74;1.01)	0.067
2005	0.76	(0.65;0.89)	0.001	0.75	(0.64;0.88)	<0.001
2006	0.72	(0.62;0.85)	<0.001	0.73	(0.63;0.86)	<0.001
2007	0.78	(0.67;0.92)	0.002	0.79	(0.67;0.92)	0.002